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National Conference on Polychlorinated Biphenyls
Nov. 19-21, 1975, Chicago, Illinois

Research Triangle Inst.

Prepared For
Environmental Protection Agency

March 1976

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NATIONAL CONFERENCE
ON
POLYCHLORINATED BIPHENYLS
(NOVEMBER 19-21, 1975, CHICAGO, ILLINOIS)

Sponsored by: Environmental Protection Agency
in cooperation with:
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Council on Environmental Quality
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ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF TOXIC SUBSTANCES
WASHINGTON, D.C. 20460

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The objectives of the conference where to bring together the latest data and best available expertise to help clarify the problems associated with the manufacture, use and disposal of PCBs ... help assess the effectiveness of steps taken to reduce the problems associated with PCBs ... provide a platform for interested parties to present previous neglected data concerning PCBs ... help clarify the feasibility and complications of steps to reduce the problems associated with PCBs.

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(November 1975, Chicago, Illinois)

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FOREWORD

The proceedings for the "National Conference on Polychlorinated Biphenyls" is the fourth report submitted under Contract No. 68-91-2928 to the Office of Toxic Substances for the Environmental Protection Agency. The three previous proceedings submitted under this contract dealt with Environmental Aspects of Chemical Use in Rubber, Well-Drilling, and Printing Operations. The PCB Conference was held at the Pick-Congress Hotel, Chicago, Illinois, on November 19-21, 1975.

The objectives of this conference were to bring together the latest data and best available expertise to help clarify the problems associated with the manufacture, use, and disposal of PCB's; to assess the effectiveness of steps taken to reduce the problems associated with PCB's; to provide a platform for interested parties to present previously neglected data concerning PCB's; and to help clarify the feasibility and complications of steps to reduce the problems associated with PCB's.

Dr. John L. Buckley, Consultant, Office of Research and Development, Environmental Protection Agency, Washington, D.C., was the General Chairman of the conference.

Mr. Thomas E. Kopp, Office of Toxic Substances, Environmental Protection Agency, Washington, D.C., was the Technical Coordinator of the conference.

Mr. Karl E. Bremer, Surveillance and Analysis Division, Region V, Environmental Protection Agency, Chicago, Illinois, was site coordinator and facilitator.

Mr. Franklin A. Ayer, Manager, Technology and Resource Management Department, Research Triangle Institute, Research Triangle Park, N.C., was the Conference Coordinator and Compiler of the proceedings.

ACKNOWLEDGMENTS

The National Conference on Polychlorinated Biphenyls owes its success to many, many people--to the organizers who arranged and accomplished such a massive undertaking, to the program chairmen and speakers who contributed so much insight and information, and most importantly, to the attendees whose interest and response made it all so worthwhile.

My special thanks to the Honorable Russell Train and the Honorable Nathaniel Reed for their supportive remarks at the Conference. Also, special thanks go to Mr. Francis Mayo, Director of EPA's Region V, Chicago; Mr. Karl Bremer of his staff; and Mr. Thomas Kopp of the Office of Toxic Substances for their superlative organizational work.

In extending appreciation to one and all, I also express gratitude to Mr. Franklin A. Ayer of the Research Triangle Institute, and his staff members Loren Clarke, Roger McGuffey, Brenda Idol, Helen Cantwell, Dianna Morgan, and Denise McCampbell for the smooth manner in which the conference proceeded, and the efficient operation of the conference facilities and support services.

John L. Buckley
General Chairman

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19 November 1975

OPENING REMARKS

John L. Buckley, Ph.D.*
General Chairman

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*Consultant, Office of Research and Development, Environmental Protection Agency, Washington, D.C.

OPENING REMARKS

John L. Buckley, Ph.D.*

I'd like to call this meeting to order. I am John Buckley, with the Environmental Protection Agency. I'd like to welcome you here to this National Conference on PCB's, with the subtitle of PCB's in the Environment in 1975.

Most of you in the room are from the United States, but there has been major participation from our Canadian neighbors to the north, and in addition, to my knowledge, representatives from France, Belgium, the Netherlands, and Japan.

I'd like to extend my personal thanks to my many colleagues in the EPA both in Washington and in the regions and in the laboratories, and my colleagues in other Federal agencies, in the industry, and in academia who were helpful in getting this conference organized in the very short period of time we had. I think almost everyone that I talked to and invited and asked to participate has agreed to do so, and I really look forward to the next 2 days as a great opportunity.

As I have gone through the process of helping to arrange the program, many questions have come to mind. Most of these are listed in the program, and I guess my aspiration is that we have some additional insights, perhaps some answers in relation to those questions before we leave.

I think there is a great opportunity for us to learn together and to go forward from here with a better common understanding and perception of the situation in regard to PCB's in the environment.

I'd ask you to remember that we're here to try and assemble and evaluate what we jointly know, and we're here also to avoid conclusions as to what it all means. It doesn't mean we need not reach conclusions, but it does mean it's probably not appropriate to try and tie everything down and understand just where we've been by the end of this meeting.

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*Consultant, Office of Research and Development, Environmental Protection Agency, Washington, D.C.

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19 November 1975

KEYNOTE ADDRESS

The Honorable Russell E. Train*

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*Administrator, Environmental Protection Agency, Washington, D.C.

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ENVIRONMENTAL PROTECTION
R_X FOR PUBLIC HEALTH

Russell E. Train*

More than 4 years ago, while I was serving as Chairman of the President's Council on Environmental Quality, a Federal interagency task force began addressing itself to the same basic question that concerns this conference: What do we know and what should we do about polychlorinated biphenyls, or PCB's?

In its report of May 1972, the task force concluded that PCB's were highly persistent, could be found in all parts of the environment, could "bioaccumulate" to relatively high levels in fish, and could have serious adverse effects on human health. The task force recognized, at the same time, that for uses in closed electrical systems, PCB's had some very real advantages over other materials. They conduct heat, but not electricity. The only available substitutes for use in capacitors and in transformers—which are widely used in indoor electrical systems—were flammable. It appeared that to ban PCB's from these uses would be, in effect, to substitute a safety hazard for a health hazard.

The task force, as a result, recommended the discontinuance of all current uses of PCB's except in closed electrical systems. It also called for early enactment of the Toxic Substances Control Act to provide the regulatory authority required not only to deal more effectively with the problems posed by PCB's and other chemicals already in the environment, but to take sensible steps to prevent such chemicals from posing such problems in the first place.

Already in 1972, the sole American producer of PCB's had voluntarily restricted the sales of PCB's to uses in closed electrical systems. Both the Food and Drug Administration and the Environmental Protection Agency announced actions designed to reduce the levels of PCB's in our food and our waters.

The United States asked the Organization for Economic Cooperation and Development (OECD) to take appropriate action, on the international level, to control PCB's. In February 1973, in the first international agreement aimed at limiting the production and use of chemicals in order to protect the environment, the member countries of the OECD announced their decision to prohibit the use of PCB's for industrial or commercial purposes except in certain closed systems. I might add that one member country, Japan, after PCB contamination of rice oil adversely affected 1,000 people, has banned the future production or import of PCB's.

We believed that all these measures added up to an effective, comprehensive program that would "take care" of the PCB problem, and would enable us to continue to take commercial advantage of the unique properties of PCB's while insulating the public and the environment against exposure to hazardous levels of these chemicals.

Instead, more than 3 years later, we find that, although PCB levels in food have steadily declined, PCB's are present in our environment to a far greater degree and at higher levels than we have previously thought. We have found high PCB levels—levels greatly exceeding FDA guidelines—in salmon, striped bass, and other fish in the Great Lakes, the upper Mississippi River, Southern California, the Gulf of Mexico and in the Hudson River and other waterways in New York State. Our selective sampling of drinking water disclosed the presence of PCB's in the water supply of two communities.

The evidence we have accumulated over the past 3 years has underscored our original concern over the toxicity of PCB's and over the potential health hazard posed by the presence of high PCB concentrations in waterways, in water supplies, and in fish. This, in brief, is the situation we find ourselves in today.

We have called this conference to help us deal with it, to determine, as I said at the outset, what we know and what we should do about PCB's.

We do know one thing that we *must* do, that we should have done 3 years ago when the task force urged us to do so, and that, had we done it, might have enabled us to really come to grips with the PCB problem and rendered this conference unnecessary—that is, *to enact an effective toxic substances control law*. I cannot help but recall that when I became the first Chairman of the Council on Environmental Quality in February 1970, my very first directive to our small staff was to develop a legislative proposal for dealing with this class of problem. The time had clearly come for an effective mechanism to deal broadly with such problems, not only after the fact, but also to help prevent their occurrence in the first place.

Almost 5 years have now passed since President Nixon first proposed such legislation to Congress. Over those 5 years we have introduced into the commercial market an estimated 600 chemical compounds annually. We have done so without any systematic, advance assess-

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*Administrator, Environmental Protection Agency, Washington, D.C.

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ment of their potential impact upon public health. Yet, as we have learned through our experience with such materials as vinyl chloride, we may not discover how harmful a compound can be until years after it has become a rather commonplace item in our everyday lives, even a significant factor in our economy. Also, again and again we find ourselves engaged in an extremely difficult and drawn-out struggle to protect the public from a hazard to which it has already been exposed while at the same time trying to avoid putting people out of business or out of work. We find ourselves trying to choose between a health hazard and a safety hazard. We find ourselves without the authority we need to really cope with the problems posed by PCB's, the authority to limit selected uses and distribution of PCB's as well as to require testing concerning the health and ecological effects of proposed substitutes.

With regard to PCB's, we will continue to address the problem as effectively as we can under existing authorities, especially under the Water Act, while at the same time recognizing the inherent inadequacies of the piecemeal approach we are forced to take. We are also working on measures we would propose under any toxic substances bill that may be enacted. They would take into account, not only the available information on toxicity and exposure levels, but also the impact of any regulatory steps upon business, employment, and the economy. I look forward to the contributions this conference can make to the development of an effective regulatory program for PCB's both under existing authority and under a toxic substances law.

About a month ago, in testimony prepared for delivery before the Environment Subcommittee of the Senate Commerce Committee, Dr. David Rall, who is scheduled to follow me this morning, said, and I quote, "Toxic substances control legislation which prevents the exposure of segments of the population to disease-producing substances is a key element of preventive medicine." There is, indeed, an increasing body of evidence, and an impressive array of expert opinion that we may be approaching the whole question of human health from the wrong side, that, as a matter of national policy as well as of personal practice, an ounce of prevention may well be worth a pound of cure.

The Department of HEW estimates that our total national health bill this year will add up to nearly $120 billion. Yet a good many informed observers believe that, because most of that money goes for cure rather than prevention, we are not getting what we pay for. Our traditional health care system, they say, simply cannot cope with environmentally-induced diseases. Dr. Ernst L. Wynder, president of the American Health Foundation, has pointed out that heart disease, cancer, stroke, and accidents account for 70 percent of deaths among Americans. And the chief causes of these diseases are "environmental" in the broadest sense of that term. "Thus," Dr. Wynder concludes, "in a society where infectious diseases have been largely overcome through sanitary measures, immunization, and antibiotics, the major causes for today's death toll are chronic diseases. This death toll is largely due to unhealthy life styles, unhealthy working environments and disease-producing products."

The more sophisticated and sensitive our monitoring devices become, and the more data we accumulate on the health effects of pollutants and other agents in the environment, the worse things look. Over a year ago, scientists uncovered disturbing evidence that children, whom we had believed unaffected in any lasting way, can contract chronic and acute disabilities as a result of air pollution. As many as 20 percent of the children in a city such as New York, one study concluded, can develop severe and chronic respiratory diseases. Another study in a southern city with relatively heavy air pollution had similar results. More recently, a group of scientists reported that the most significant factor in a dramatic drop in deaths in the San Francisco area during the gas shortage early last year—a 13.4 percent decrease in deaths compared with the same period over the previous 4 years—was reduced exposure to pollutants from auto exhausts.

We are spending around $1 billion this year on research into cures and causes of cancer. The National Cancer Institute has estimated that the actual cost of cancer to people amounts to tens of billions of dollars a year. Yet the World Health Organization estimates that from 60 to 90 percent of all cancer is the result of "environmental" factors, again, in the broadest sense of that term. We have all read the news stories recently concerning the 1975 Nobel award to three American scientists for research into possible links between viruses and cancer. I was struck by the fact that, in their first public statements upon receiving the award, two of these scientists stressed the fact that, in the words of one of them, Dr. David Baltimore of the Massachusetts Institute of Technology, "the role of viruses in cancer is small' and that " the best hope today for cures is research into environmental causes of cancer." "This," Dr. Baltimore went on to say, "is a good place to put funds now."

We should understand, as well, that while environmental protection often appears to involve substantial costs, we really have no choice about whether or not we are going to bear these costs. Society has already been bearing these costs in one form or another—in the loss of recreational uses of rivers and beaches; in the increased treatment costs of our drinking water; in the damage from air pollution to buildings, farm crops, and forests; and most importantly, through medical and hospital bills, time lost on the job because of illness, human suffering, earlier mortality, and the like. When we control and cut pollution at the *source*, we are shifting its costs from the shoulders of society as a whole onto those of the polluter, where they belong in the first place. Such costs then tend to be passed on to the polluter's customers. But this is the most efficient way of allocating these costs and of encouraging, at the same time, the development of both processes and practices that generate less pollution. Moreover, all our experience indicates that the cost of the particular pollution to society as a whole is usually far greater than the cost of cleanup and control. We estimate, for example, that measurable annual damages of $11.2 billion from particulates and sulfur oxide are more than twice the annual costs of control.

What all of this suggests to a layman such as myself is that both our popular understanding of, and our public approach to, health care and disease control are going to have to undergo a searching reexamination and, I suspect, radical revision. It suggests that, some of our most effective "health care" dollars, at least when they are well spent, may be the "disease prevention" dollars we spend to curb and control pollution and other agents that we introduce into our own environment. It suggests that the battle against disease must increasingly be fought, not simply in the hospitals and the doctors' offices, but in our streets and our homes and our offices, in our air and our water, in our food and our products, in our personal habits and lifestyles. It suggests that if, in the words of Dr. Irving Selikoff of the Mount Sinai School of Medicine in New York, "environmental disease is becoming the disease of the century," then environmental protection, in the broadest sense of the phrase, must become the most important ingredient in any national health program.

Such a broad prescription may seem far removed from the more immediate and urgent concerns of this conference. But I think we all understand that it is our failure to get at the real roots of the problem that concerns us here, and our preoccupation instead with the symptoms and surfaces and single instances of things that has made this conference and the problem it addresses so critical.

At the start of my remarks, I successfully resisted the temptation to issue the dramatic announcement that this was my last PCB's conference. It is, however, my fervent hope that you do your work so well at this conference that we will never need to call another one. I thank you for coming, and I look forward to seeing the results of your work.

19 November 1975

Session I:

HEALTH EFFECTS AND HUMAN EXPOSURE

David P. Rall, M.D., Ph.D.*
Session Chairman

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*Chairman, Department of Health, Education, and Welfare Committee to Coordinate Toxicology and Related Programs, and Director, National Institute of Environmental Health Sciences, National Institute of Health, Research Triangle Park, North Carolina.

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INTRODUCTORY REMARKS

David P. Rall, M.D., Ph.D.*

The history of PCB's is a remarkable case study in the continuing story of toxic substances and the public health. From 1929, when it was first manufactured in the United States, until 1966, it was presumed that this substance was being used primarily in a closed environment. However, in 1966 PCB's were found in fish in the Baltic Sea. Then they were discovered in birds and other animals. Since then it has been demonstrated they have an almost global distribution. Three years after being discovered as an environmental contaminant, they were determined to be the causative agent in an outbreak of a disease in Japan now called "Yusho" or "oil" disease.

Thus, in a relatively short period of time, we saw a manmade compound introduced into commerce for a relatively narrow purpose, a closed use, broadened through new applications, and discovered to be hazardous to animals, birds, fish, and to man. In slighly more than two generations, we had closed the all-too-frequent circle of progress: product development, distribution, use, and resulting hazard to human health.

In 1971, we became concerned that the PCB's appeared to be an ever-expanding problem whose potential limits were essentially unknown. Thus, in December of 1971 we called a 2-day open meeting to discuss the entire range of current and potential health problems associated with the widespread use and dispersion of PCB's. The meeting was broadly attended by scientists and administrators throughout the world who were concerned with this problem.

The scientists at that meeting focused on the entire spectrum of problems that needed investigation. Their papers ranged from biological concerns: animal toxicology, mechanisms of action, and human body burden; to regulatory and control concerns: environmental transport, distribution, and alteration, and occurrence. In his "Where do we go from here" summary, Dr. Norton Nelson singled out two general areas of concern.(ref.1).

First, relative to environmental distribution patterns, including: (1) further refinement of sources, and (2) better quantitation of discharge amounts by route into water and air. Second, he pointed out that there were inadequate data to determine if the PCB's caused malignant tumors in laboratory animal studies and urged that we follow the ongoing studies with care. He also pointed out the potential deleterious effects of the PCB's on reproduction in mammals.

In following up these and the other points Dr. Nelson made, scientists, many of them in DHEW, have been engaged in attempting to develop the information required for the resolution of the biological aspects of this problem. Last month, in order to insure that our Department's efforts are as effective as possible, we established a Subcommittee on PCB's of the DHEW Committee to Coordinate Toxicology and Related Programs.

Through this Subcommittee we will:

1. Assemble, review, and interpret data that assess the health significance of polychlorinated biphenyls, and
2. Formulate recommendations as to future research needs.

In all of these efforts, we will be cooperating with those agencies which must carry out regulatory responsibilities in this difficult area. Continuing collaborative efforts by biological scientists here and abroad have brought us far toward the resolution of these problems. The reports which you will hear relating to Health Effects and Human Exposure in the session which follows should indicate the progress which we have made since the 1971 conference.

REFERENCE

1. Norton Nelson, "Comments on Research Needs," *Environmental Health Perspectives 1* Vol. 1 (April 1972), pp. 181-185.

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SOME OF THE RECENT FINDINGS CONCERNING YUSHO

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Abstract

Analysis of Yusho disease, which was first detected in 1968, has been limited and analyses have produced varying results. Yusho oil has been determined to contain a high level of polychlorinated dibenzofurans (PCDF's). Nagayama et al. found that PCDF levels in the oil were especially high when the oil was contaminated with PCB's used as a heat transfer medium. Their studies also showed that the concentration of PCDF's is much closer to that of PCB's in liver than in adipose tissues in patients with Yusho.

The current clinical state of patients with Yusho is dicussed at length. Subjective symptoms, dermatological findings, serum triglyceride levels, liver conditions, mortality rates, and the effects on children born to mothers with Yusho are all reported.

INTRODUCTION

More than 7 years have passed since the outbreak of an epidemic of Yusho in 1968. According to the latest tabulation, Prof. Omae, current chief of the Study Group for the Therapy of Yusho, reported that a total number of 1,291 patients have been registered as Yusho in 22 prefectures of Western Japan by April 30, 1975 (ref. 1).

We would like to describe some of the recent findings concerning Yusho which we think to be particularly relevant for understanding of toxicity of PCB's. For more detailed information, readers are advised to refer to original papers appearing mainly in the fourth and fifth reports of the study on Yusho and PCB (ref. 3). Most of the patients described in these reports are those living in Fukuoka prefecture. Published information of patients living in other areas are unfortunately very few, so with a few exceptions no reference will be made to them.

PCB's IN THE BODIES OF PATIENTS WITH YUSHO

1. *Tissues*

First of all, the concentration of PCB's retained in the tissues and fluids of patients with Yusho should be referred to before their current clinical state will be discussed.

Although no accurate estimation is feasible because of the very limited number of analyses so far made, the concentration of PCB's in adipose tissues of patients seems to have been fairly high soon after the occurrence of poisoning—that is, in November 1968, at least 1 month after the discontinued use of the toxic rice oil by patients, as shown in table 1. The corresponding concentrations were considerably lower in 3 patients who died in the next year, 1969. However, no such marked difference could be seen between those who died in 1969 and the subsequent decedents, although cases 7 and 10, who died in 1970 or 1975, showed quite low levels of PCB's in adipose tissues. As compared with the figures available from a nationwide survey on residual PCB's in autopsied tissues, the levels noted in the recent decedents are considered to be fairly close to the usual level of ordinary autopsied materials. Similar facts were also noted for PCB concentration in the skin and liver.

2. *Blood*

Since the analysis of PCB's in blood started only after 1972, no figures are available in regard to the blood levels of PCB's in patients in the earlier stage of poisoning. As shown in table 2, however, it is clear that the blood levels of patients had approached the level of ordinary persons already in 1972, although they were still significantly higher than that level. This fact as well as the previous fact of lowered tissue levels of PCB's in recent decedents seem to be rather surprising if we consider also that the majority of patients are still showing various clinical symptoms, as will be discussed later.

3. *Gaschromatographic Patterns of PCB's in the Bodies of Patients*

Masuda first noted a peculiar common gas-chromatographic pattern of PCB fractions isolated from various tissues, blood, and breast milk of patients with Yusho and called the attention of the Study Group for the Therapy of Yusho to it in early 1972. Figure 1 shows a typical example of such a pattern in compariso

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Table 1. PCB's concentration in tissues of patients with Yusho and other diseases

Case	Time of death, operation	PCB's (ppm)						Reference
		Skin		Adipose tissue		Liver		
		Whole basis	Fat basis	Whole basis	Fat basis	Whole basis	Fat basis	
Case 1 High school boy	Nov. 1968			76 (face) 13 (abdomen)				
Case 2, 3 Adult male, female	Nov. 1968			32, 46 (cheese-like substance from acneform eruptions)				10
Case 4 Boy, 13 yr	July 1969			1.3 (mesentery)	3.7	0.14	9.5	
Case 5 Male, 25 yr	July 1969	1.2	8.7	2.8 (mesentery)	15.1	0.2	10.4	
Case 6 Male, 73 yr	Nov. 1969	1.0	4.4	3.8 (mesentery)	8.4	0.07	3.1	11, 12
Case 7 Female, 48 yr	Dec. 1970	0.6	0.8	0.7 (mesentery)	0.9	0.07	1.3	
Case 8 Male, 46 yr	May 1972	1.8	3.2	4.3	6.5	0.08	8.4	
Case 9 Female, 33 yr	Sept. 1972			1.9 (subcutaneous)	2.9			
Case 10 Male, 72 yr	April 1975			0.19 (mesentery)	0.4	0.04	3.0	14
National survey Males and females 25 - 49 yr	1973	0.04 - 1.7 (n = 54)		0.2 - 4 (n = 47)	0.3 - 6.4 (n = 48)	0.01 - 0.6 (n = 51)	0.02 - 3.1 (n = 30)	13

Table 2. PCB's in blood patients with Yusho, workers and ordinary persons

Material	No. of subjects	Time of examination	PCB's (ppb) Whole basis mean ± S.D.	Range	Reference
Whole blood					
Yusho patients	41	March - August 1973	7	2-26	7
Ordinary persons	37	1972	3	1-7	
Plasma					
Yusho patients	15	Jan. 1972	6.3 ± 4.0	2-15	8
Normal persons	82		3.0 ± 1.3	1- 7	
Whole blood					
Yusho patients	25		4.8 ± 2.9	1-12	
Normal persons	11		2.8 ± 1.5	1- 6	
Whole blood					
Workers[a]	23	1972	364 ± 262	60-920	9

[a] Workers engaged in the production of Kanechlor 200-600 in the air containing 0.05 to 0.2 mg/m^3 of PCB's [Kanechlor-300 + Kanechlor-400 (1:1)]. Two of them showed dermal signs.

with the common one seen in ordinary persons, which is very much close to that of a mixture of Kanechlor 500 + 600 (1 : 1). It is easily notable that the peak 1, which appears immediately after p,p'-DDE in the gas chromatogram in figure 1, is very low in patients with Yusho as compared with ordinary persons, while the peak 5 is much more prominent in Yusho than in ordinary persons. Masuda and his associates observed this peculiar pattern (designated as type "A") in blood of about 60 percent of patients with Yusho, and a somewhat similar pattern (designated as type "B") in about 37 percent (refs. 4,7,11,12).

Takamatsu et al. also observed the same peculiarity in the blood of patients with Yusho (ref. 8). It was further demonstrated that those patients who show the peculiar gas-chromatographic pattern as designated as pattern A by Masuda et al. have a higher average concentration of PCB's in their blood than do other patients (refs. 4,7). Abe et al. (ref. 23) examined in 1974 the PCB residues in the plasma of 30 children born to 18 mothers who had consumed Yusho oil at Goto Islands, Nagasaki prefecture. The specific gas-chromatographic pattern of type A was seen among 24 percent of the children and 44 percent of their mothers, but the peculiarity seemed somewhat less marked as compared with the one seen among patients in Fukuoka prefecture.

The chemical and toxicological nature of the compound or compounds yielding the above-mentioned peak 5 must be clarified but have not yet fully been examined. Masuda, however, considers the peak 5 to be 3,4,2',3',4',5'-hexachlorobiphenyl, judging from its retention time on the Apiezon L column developed by Jensen and Sundströn (ref. 22).

A : FATTY TISSUE OF YUSHO PATIENT
B : FATTY TISSUE OF ORDINARY PERSON
C : KANECHLOR 500 + 600 (1:1)

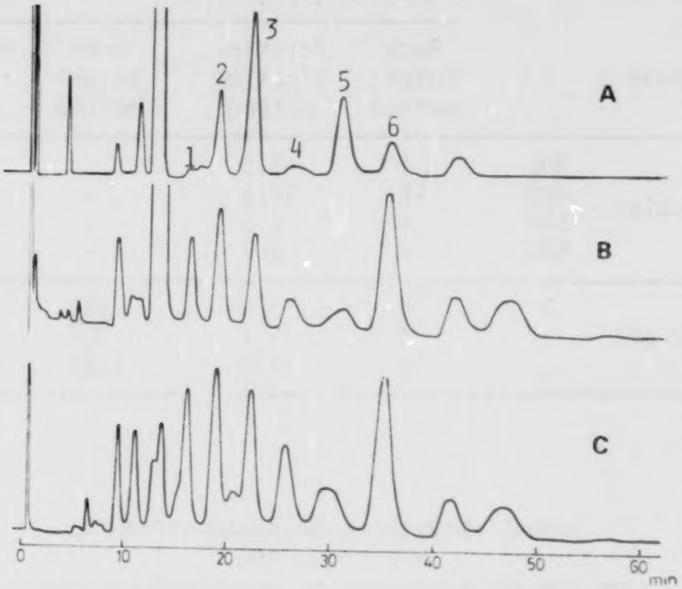

Figure 1. Gaschromatograms (ECD) of PCB's on SE-30.

POLYCHLORINATED DIBENZOFURANS IN KANECHLORS, "YUSHO OIL," AND TISSUES OF PATIENTS

1. *Polychlorinated Dibenzofurans in Kanechlors and "Yusho oil"*

Recently Nagayama et al. analyzed Kanechlors and three samples of toxic "Yusho oil" used by three independent families with Yusho for polychlorinated dibenzofurans (PCDF's) and polychlorinated dibenzo-p-dioxins (PCDD's) (ref. 16). They did a column chromatographic fractionation of PCDF's and PCDD's from a bulk of PCB's by using activated alumina as adsorbent, and n-hexane, n-hexane containing 20 percent carbon tetrachloride, or n-hexane containing 20 percent methylenchloride as eluent. The fractions thus obtained were subjected to gas-chromatographic and mass-spectrometric examination. Quantitative estimation of PCDF's and PCDD's was made by two methods, namely by measuring the gas chromatographic peak heights and by measuring the gas chromatographic peak area of perchlorinated derivatives of these compounds.

Although no PCDD's were found in any of these samples, PCDF's were found in all of them. As shown in table 3, KC-400 contained the highest concentration of PCDF's, about 18 ppm of PCDF's consisting of dichloro- up to pentachloro- dibenzofurans. A peak with the same retention time as that of 2,3,7,8-tetrachlorodibenzofurans, which was kindly provided by Dr. J. G. Vos, was noted in its gas chromatogram. All of the three samples of "Yusho oil" also contained about 5 ppm of PCDF's, the major constituents of which were tetra- and pentachlorodibenzofurans. Here again, the peak with the same retention time as 2,3,7,8-tetrachlorodibenzofuran was noted. Kashimoto also found 1.6 ppm of PCDF's in another batch of "Yusho oil," as shown in table 5 (ref. 18). Table 4 summarized the concentrations of PCDF's in Kanechlor-400, reported by several authors. As clearly noted, there is a fairly large discrepancy in their findings. Whether or not it was due to their analytical procedures or to batch difference cannot be decided at the present time.

Another important fact was disclosed by Nagayama et al. They determined the concentration of PCB's in "Yusho oil" as approximately 1,000 ppm or slightly less than 1,000 ppm, as shown in table 3. Since "Yusho oil" is known to have been contaminated with Kanechlor-400, the ratio of the concentration of PCB's to that of PCDF's in "Yusho oil" is expected to be about 1,000:0.018. The observed ratio, approximately 1,000:5,

Table 3. PCDF's and PCB's in Kanechlors and Yusho oil

Sample		PCDF's (ppm)		PCB's (ppm)	
		Peak height method	Perchlorination method	Peak height method	Perchlorination method
Kanechlor	300	1	1.5	-	-
	400	18	16.6	-	-
	500	4	2.5	-	-
	600	5	2.7	-	-
Yusho oil	A	5	4.4	830	870
	B	4	5.1	900	920
	C	5	5.2	1030	980

Table 4. Reported concentrations of PCDF's in Kanechlor-400

Authors	Year	Concentration (PPM)
Roach et al.[a]	1974	1
Nagayama et al.[b]	1975	18
Kashimoto et al.[c]	1975	33

[a] Ref. 17.

[b] Ref. 16.

[c] Ref. 18.

was thus about 250 times higher than expected (table 5). The reasons for this great discrepancy are not clear yet. It should be noted, however, that the sample of Kanechlor-400 analyzed was an "unused" one, while the Kanechlor-400 present in "Yusho oil" was "used" as heat transfer medium. This fact suggests a practically important possibility that Kanechlor-400 and probably other commercial PCB's, too, will increase their PCDF concentrations when used as heat transfer medium.

2. *PCDF's in Tissues of Patients with Yusho*

Nagayama et al. (ref. 24) further examined the tissues of patients with Yusho for their possible content of PCDF's. Table 6 summarized their findings. Adipose tissues and liver from two ordinary persons who died of accidents contained no detectable amount (< 0.1 ppb) of PCDF's, but those from three patients with Yusho who died in 1969 or 1972 were all shown to contain PCDF's. Figures 2 and 3 show their gas-chromatograms and mass spectra. The concentration on a whole basis was 0.009 ppm on average for adipose tissues and 0.013 ppm for liver. This seems to be a rather surprising fact because in the case of PCB's the concentration on a whole basis is usually much lower in liver than in adipose tissues. When compared on fat basis, another interesting fact was noted that PCDF concentration was much higher in liver than in adipose tissue. Although the number

Table 5. Concentrations of PCB's and PCDF's and their ratios in various materials

Materials		PCB's (ppm)	PCDF's (ppm)	PCB's / PCDF's	Reference
Kanechlor-400		1,000,000	ca. 20	50,000	16
Yusho oil	A[a]	ca. 1,000	5	200	
	B[b]	134	1.6	84	18
Patient with Yusho	Adipose tissue	1.3	0.009	144	24
	Liver	0.05	0.013	4	

[a] Samples of the rice oil produced on February 5 or 6, 1968.

[b] A sample of the rice oil produced on February 10, 1968.

Table 6. PCB's and PCDF's in tissues of patients with Yusho and ordinary persons

Subjects	Tissue	Case No.	Time of death	PCB's (ppm)		PCDF's (ppm)		Ratio PCB's/RCDF's	
				Whole basis	Fat basis	Whole basis	Fat basis	Whole	Fat
Yusho patients	Adipose	1	1969	1.4	3.4	0.013	0.03	108	113
		2	1969	1.3	8.5	0.006	0.04	217	213
		3	1972	1.2	2.1	0.007	0.01	171	210
		Avg.		1.3	4.7	0.009	0.003	144	157
	Liver	1	1969	0.05	4.7	0.025	2.3	2	2
		2	1969	0.06	5.6	0.010	1.1	6	5
		3	1972	0.03	3.5	0.003	0.3	10	12
		Avg.		0.05	4.6	0.013	1.2	4	4
Ordinary persons	Adipose	1	1975	1.0	1.4	ND	ND		
		2	1975	0.4	0.7	ND	ND		
	Liver	1	1975	0.08	1.3	ND	ND		
		2	1975	0.02	1.0	ND	ND		

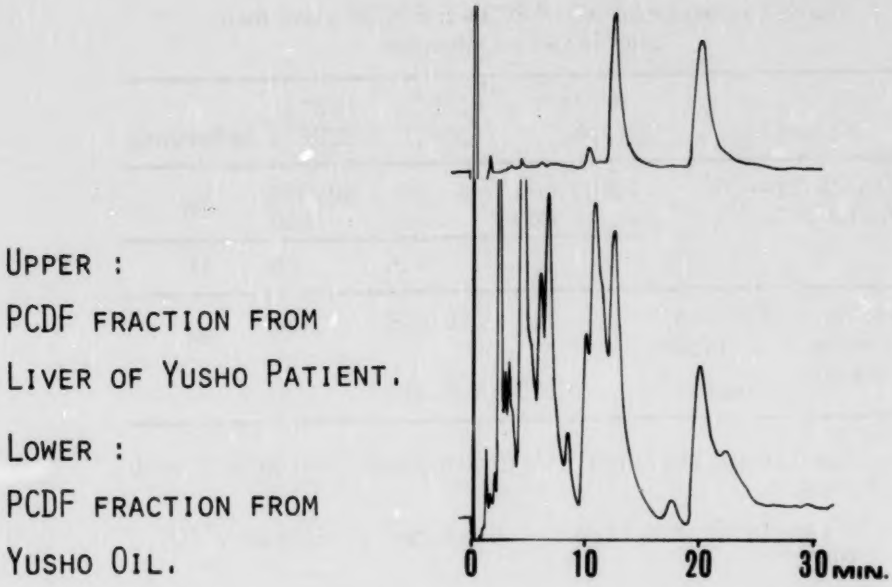

Figure 2. Gaschromatograms of PCDF fractions from liver of patient with Yusho and from "Yusho oil."

of analyses made is quite limited and nothing can be said with certainty, this fact seems to deserve attention. As shown in table 6, these different behaviors in tissue distribution of the compounds caused a remarkable difference in ratio of PCB's to PCDF's between adipose tissues and liver. It was thus demonstrated that the concentration of PCDF's is much closer to that of PCB's in liver than in adipose tissues in patients with Yusho. The PCDF's identified in liver were mainly penta- and hexachlorodibenzofurans, containing only a trace of tetrachloroisomers.

CURRENT CLINICAL STATE OF PATIENTS WITH YUSHO

In 1974, Prof. Urabe (ref. 2), former chief of the Study Group, reported that the dermal and mucosal signs that were most marked at the incipient stage of the poisoning had gradually been improved, while symptoms such as general fatigue, poor appetite, in constant abdominal pain, heavy headedness and headache, feeling of numbness and pain at the limbs, and cough and expectoration of sputum, all of which are considered to be due to some internal disturbances, have become more prominent year by year. In view of these tendencies together with the discovery of a characteristic gas-chromatographic pattern of PCB's remaining in the blood and tissues of patients, the diagnostic criteria for Yusho was revised in 1972 (ref. 2), as shown in table 7. As compared with the former one, the revised criteria describe briefly the dermal and mucosal lesions but newly refer to other noncutaneous objective signs and findings from several laboratory tests. It should be noted, however, that the new criteria do not refer to any specific liver function tests.

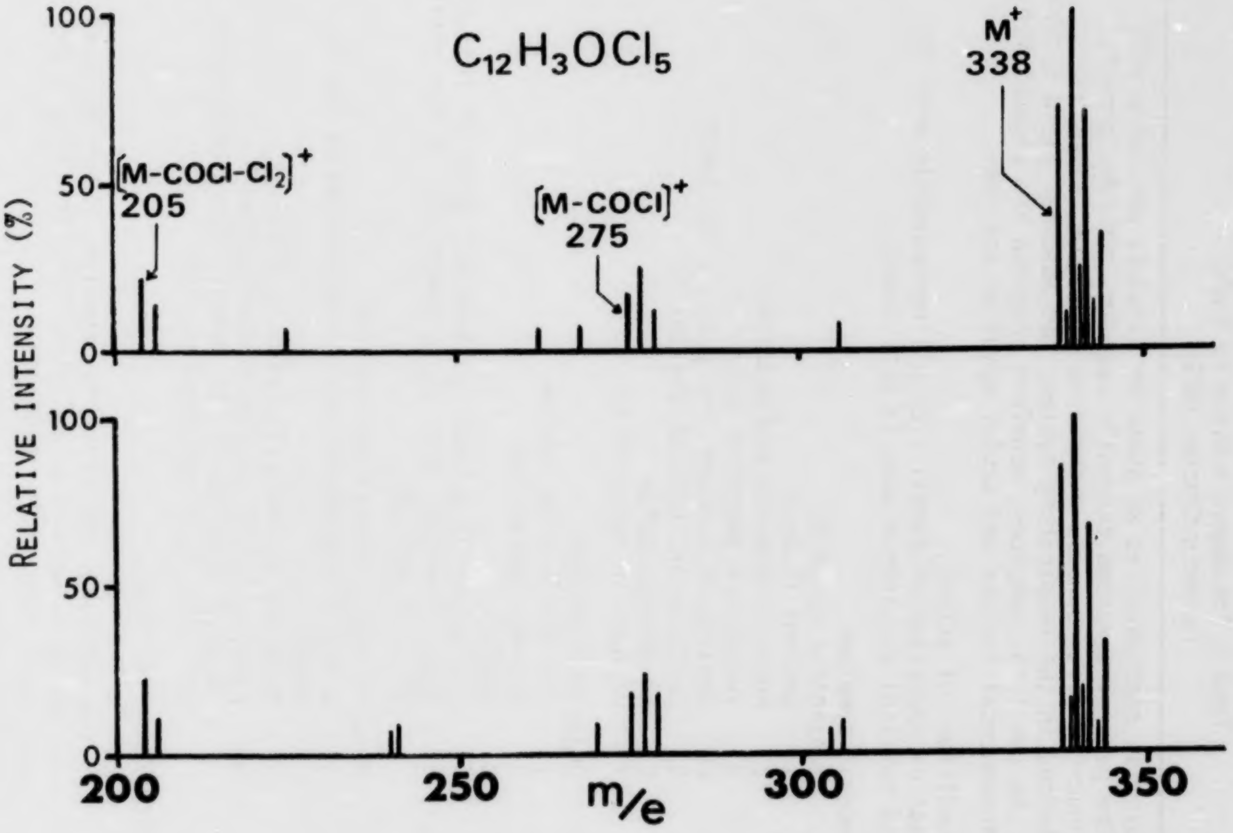

Figure 3. GC-MS of PCDF's fraction from liver of patient with Yusho (upper) and of synthesized PCDF's (lower).

Table 7. The diagnostic criteria for Yusho (revised in October, 1972).

Yusho is considered as an acute or subacute poisoning with PCB. The general symptoms currently seen are retarded growth, neuroendocrine disturbances, phenomenon of enzyme induction, disturbances in the respiratory system, and abnormal lipid metabolism. As the local symptoms, acneform eruption and pigmentation as cutaneomucosal lesions and ocular symptoms are seen.

1. Conditions of attack
   Fact of ingestion of Kanemi rice oil contaminated with PCB and familial occurrence seen in most cases.
2. General symptoms
   a. Subjective symptoms
      1) general fatigue
      2) heavy headedness and headache
      3) inconstant abdominal pain
      4) feeling of numbness and pain at the limbs
      5) swelling and pain at the joints
      6) cough and sputum
      7) changes in menstruation

   b. Objective symptom
      1) bronchitis-like symptom
      2) sensory neuropathy
      3) bursitis
      4) inhibition in growth and abnormal teeth in children
      5) Small-For-Dates baby and pigmentation of the entire skin of newborns

   c. Results from clinical examination
      1) abnormal properties and concentration of PCB in blood
      2) increase of neutral lipids in blood
      3) anemia, lymphocytosis, hypoalbuminemia
      4) reduced velocity of the sensory nerve conduction and adrenocortical hypofunction
3. Cutaneomucosal signs
   a. Acneform eruption
      Black comedones and acneform eruptions which are seen at the face, buttocks, and other intertriginous sites and their suppurative tendency.

   b. Pigmentation
      Pigmentation of the face, palpebral conjunctiva, gingiva, and nails of the fingers and toes.

   c. Ocular signs
      Swelling and hypersecretion of the Meibomian gland and palpebral edema.

(Translation was made by Kuratsune)

1. *Subjective Symptoms*

Table 8 shows that a considerable portion of the patients are still suffering from various subjective symptoms in recent years. Kōda and Masuda examined their possible association with PCB concentrations in blood, finding no positive association at all (ref. 4). Umeda also reported on various symptoms due to disturbances of higher nervous activities (e.g., forgetfulness) complained of by most patients, but no definite association between such symptoms and the blood levels of PCB's was observed (ref. 5).

2. *Dermatological Findings*

Kōda and Masuda examined 72 patients with Yusho for dermatological signs and PCB levels in the blood from April 1973 to March 1974 (ref. 4). As shown in table 9, the majority of patients were still suffering from skin lesions such as pigmentation, deformation of nail, and hypersecretion of Meibomian gland even 5 years after the poisoning. They also demonstrated another important fact that group A, consisting of patients whose blood shows the gas-chromatographic pattern A, had significantly higher prevalences of dermatologic signs such as pigmentation, acneform eruption, and deformed nails than did group B, which consisted of patients showing no such typical gas-chromatographic pattern.

Since group A had a higher average concentration of PCB's than group B, the dermal lesions seen among current patients, contrary to the subjective symptoms, seem to be causally associated with the current level or pattern of PCB's remaining in their blood. However, no conclusion could be readily made in this regard. First of all, the current excess of PCB's in the blood of patients is not remarkable in degree and is almost negligible as compared with the enormous elevation seen among the occupationally exposed workers, who nevertheless showed a rather low prevalence of dermal symptoms (table 2).

It seems rather hard, therefore, to explain the persisting dermal lesions by elevated PCB levels in blood alone. The chemical peculiarity of such PCB's and the presence of PCDF's in the bodies of current patients seem to be particularly important in this connection. However, our present knowledge does not allow us to continue discussion of the matter along this line without speculation. Furthermore, an entirely different explanation might also be possible. The skin lesions currently seen may merely be the persisting original skin lesions, the severity of which must have been determined primarily by the amount of intake of PCB's; such intake must in turn be reflected by the current PCB levels in blood of patients. According to this explanation, the

Table 8. Frequency of subjective symptoms complained by patients with Yusho from 1973 to 1974

Symptoms	Proportion[a] %
Fatigue	51.4
Headache	41.7
Phymata in articular region	8.3
Fever	2.8
Cough and sputum	56.9
Digestive disorder	40.3
Numbness of extremities	33.3
Menstrual disturbance	26.9 (7/26)

[a] Calculated by Kuratsune from original figures published by Kōda and Masuda (ref. 4).

Table 9. Prevalence of dermatological and other signs among patients with Yusho from April 1973 to to March 1974, in connection with concentration and gaschromatographic pattern of PCB's in blood

Signs		Prevalence (%)[c]			
		Group A (43 cases)	Group B (26 cases)	Group C (33 cases)	Total (72 cases)
Pigmentation	Skin	51.2[b]	0	0	30.6
	Palpebra	72.1[b]	19.2[b]	0	50.0
	Gingiba	95.3[b]	57.7[b]	66.6	80.6
	Nail	74.4[b]	34.6[b]	0	56.9
Acneform eruption		34.9[b]	0[b]	0	20.8
Comedo		34.9	23.1	0	29.2
Infection of skin		32.6	11.5	0	23.6
Deformation of nail		65.1[a]	38.5[a]	0	52.8
Alopecia		0	3.8	0	1.4
Disorder in teeth		18.6	7.7	0	13.9
Dypersecretion of Meibomian gland		93.0	80.8	100.0	88.9
PCB's in blood	Conc. (ppb) Avg. ± S.D.	7.2 ± 4.9	4.3 ± 3.1	1.7 ± 0.2	5.9 ± 4.5
	Pattern	A[d]	B[d]	C[d]	

[a] Significant ($P < 0.05$) difference.

[b] Significant ($P < 0.01$) difference.

[c] Calculated by Kuratsune from figures published by Kōda and Masuda (ref. 4).

[d] "A" means the characteristic gaschromatographic pattern of PCB's remaining in the body of most patients with Yusho. "B" means gas chromatographic patterns somewhat similar to "A". "C" means patterns indistinguishable from those of normal persons.

observed association of the dermal lesions with current blood levels of PCB's is considered as a phenomenal one but not as a causal one. In order to evaluate these different possibilities, it seems essential to examine chemically and toxicologically the PCB's and PCDF's still remaining in patients' bodies.

3. *Serum Triglyceride*

One of the most dominant objective signs seen at the incipient stage of Yusho was a markedly increased concentration of serum triglyceride. Okumura and his associates reported recently the results of their extensive followup study on 40 patients who were examined for serum triglyceride at least once a year successively for 6 years from 1969 to 1974 (ref. 19). As shown in table 10, a group of 14 male patients has shown no significant change in serum triglyceride levels since 1969, still maintaining levels as high as 160 ± 118 mg/100 ml even in 1974. For 26 female patients, however, a significant decrease was seen in 1973 and 1974 when compared with the levels in the previous years. However, 42 percent of them still showed higher levels than 110 mg/100 ml in 1974.

Okumura et al. (ref. 20) examined the possible association between serum triglyceride levels and PCB concentrations in blood in patients. As shown in table 11, they observed a significantly higher mean level of serum triglyceride in a group of patients who showed the characteristic gas chromatographic pattern A, as compared with other patients who did not show such a typical pattern. They also observed a significantly positive correlation between serum triglyceride levels and PCB concentrations in blood ($r = 0.485$).

Table 10. Results of followup study on serum triglyceride levels in patients with Yusho[A]

Subjects	PCB's pattern	No. cases	Age mean	PCB's in blood (ppb)	Triglyceride mg/100 ml	Reference
Patients	A	20	31.9	8.6 ± 5.2[b]	134 ± 60.0[a]	20
with	B	14				
Yusho	C	2	21.4	3.8 ± 2.2[b]	91 ± 39.8[b]	
Controls	C	37	34.5	2.8 ± 1.6		
Controls					74 ± 29	19.21

[a] $P < 0.05$.

[b] $P < 0.005$.

Table 11. PCB's concentrations in blood and serum triglyceride levels in patients with Yusho in 1973

Patients			Triglyceride (mg/100 ml) Mean ± S.D.					
Sex	Age	No.	1969	1970	1971	1972	1973	1974
Male	11 - 73	14	159 ± 57	166 ± 55	169 ± 60	174 ± 69	164 ± 68	160 ± 118
Female	7 - 59	26	155 ± 75	161 ± 70	155 ± 80	153 ± 63	129 ± 50[b]	111 ± 56[b]

[a] Cited from a report by Okumura et al. (ref. 19).

[b] Significantly lower than in 1969, 1970, 1971, and 1972 ($P < 0.05$).

Here again, a similar question can be raised in regard to such observed correlation, as already discussed in connection with the dermal lesions. Are the current elevated levels of PCB's in blood and their peculiarities in gas-chromatographic patterns causally connected with the abnormally high serum triglyceride levels found in patients? Since, as mentioned, the female patients started to decrease in serum triglyceride concentration in recent years, a followup examination of PCB's in their blood might give a good clue to answer the above question.

4. *Liver of Patients with Yusho*

Both PCB's and PCDF's are well-known toxic agents to the liver. PCDF's seem to be particularly toxic because a single oral administration of PCDF's as small as about 1 mg/kg could kill rabbits by severe liver necrosis (refs. 28,29). Therefore, it is reasonable to expect that patients with Yusho would have a severe liver damage. Okumura et al. (ref. 30) performed detailed medical examinations on 24 patients soon after the onset but, unexpectedly, obtained no objective findings to indicate definite liver disorders. No patients presented jaundice and only three of them had palpable livers. However, an electron microscopic examination of liver biopsy specimens conducted on a patient in February 1969 revealed a marked hypertrophy of smooth endoplasmic reticulum, indicating stimulated enzyme induction in the liver (ref. 31).

Okumura examined 38 patients with various subjective symptoms for serum enzymes, including isozymes from 1971 to 1972 (ref. 32). An increase in a fraction of lactate dehydrogenase (LDH-5) and high titers in thymol turbidity tests were observed in some of the severe cases but no definite evidence for liver disorders was obtained. Recently Hirayama et al. (ref. 33) examined 121 adult patients with Yusho and 257 healthy adult controls for serum bilirubin, demonstrating a significant lower average concentration in the patient group than in the control. They also showed significantly negative correlations between serum bilirubin and blood PCB's in concentration ($r = -0.349$, $p<0.025$) and similarly between serum bilirubin and serum triglyceride ($r = -0.215$, $p<0.05$). They considered that a lowered concentration of serum bilirubin in patients seemed mainly due to an accelerated bilirubin disposal from the blood.

Hirayama et al. investigated 125 patients for Australia antigen and antibody by the immunoelectroosmophoresis in 1971 (ref. 34). The antigen was positive in three, while the antibody was negative in all of them, indicating no difference at all in the prevalences between the patients and healthy controls. This finding seems to be important in connection with the future risk of cancer which patients might experience.

In view of all these findings, liver function tests currently available do not readily detect serious liver lesions in the patients, but it is highly desirable that adequate caution will continuously be paid to this well-known target organ of chlorinated hydrocarbons.

CHILDREN BORN TO MOTHERS WITH YUSHO

The birth of unusual babies from mothers who took "Yusho oil" during pregnancy is already well known (refs. 25,26). Their clinical features were dark brown pigmentation of the mucous membrane and the entire skin, gingival hyperplasis with pigmentation, a tendency to be small for the date, eruption of teeth at birth, hypersecretation of the Meibomian gland, and edema of the orbital area. Pigmentation of the skin disappeared in 2 to 5 months, followed by growth similar to that of normal babies.

It seems noteworthy, however, that babies with the dark brown pigmented skin continued to be born for a few years after the intake of "Yusho oil" was discontinued by mothers. Yoshimura reported on nine babies with such skin who were born to mothers with Yusho in Nagasaki prefecture from 1969 to 1972 (ref. 27). Three of such babies had been delivered by a patient from 1969 to 1971. Abe et al. (ref. 23) recently reported on PCB levels in the plasma of 30 children (aged 0–7) born to 18 mothers with Yusho in Nagasaki prefecture. Their examination was made in 1974. As shown in table 12, the PCB levels of these children were significantly higher than those of ordinary children but lower than the levels of their mothers. Their gas-chromatographic patterns of PCB's in plasma were already referred to earlier in this paper. Children fed on breast milk from mothers with Yusho tended to show a higher plasma concentration of PCB's than those who were not fed on such milk.

Yoshimura also reported an interesting case, where a baby was thought to have suffered from Yusho due exclusively to intake of PCB's through breast milk from a woman with Yusho (ref. 27). Very few data are available in regard to the concentration of PCB's in breast milk of mothers with Yusho. Masuda et al. found 0.03 – 0.06 ppm of PCB's in 5 samples of breast milk collected from a woman with Yusho within 5 days after delivery in 1973 (ref. 12). Masuda also found about 0.03 ppm of PCB's in another sample of breast milk collected a few days after a woman with Yusho delivered a baby with no dermal signs (ref. 14). The PCB levels in breast milk from patients with Yusho were therefore just within the normal range. However, the gas-chromatographic patterns of these samples were quite unique, the same as that characteristic for Yusho.

Table 12. Concentration of PCB's in plasma of children born to mothers with Yusho

Subjects	Subjects	PCB's in plasma (ppb) Range	Mean ± S.D.	Reference
Mothers with Yusho	18	3-33	11.2 ± 7.32	
Children born to above mothers	30	1-20	6.7 ± 4.28	23
Ordinary children	14	1-8	3.7 ± 1.97	

Table 13. Deaths seen among patients with Yusho[A]

Cause of death	Number
Malignant neoplasms	9
Stomach cancer	2
Stomach cancer + liver cancer	1
Liver cancer + liver cirrhosis	1[b]
Lung cancer	1
Lung Tumor	1
Breast cancer	1
Malignant lymphoma	2
Cerebrovascular lesion	3
Amyloidosis	1[b]
Osteodystrophia fibrosa	1[b]
Myocardial degeneration + pericarditis	1[b]
Status thymicolymphaticus	1[b]
Liver cirrhosis	1
Suicide	1
Senility	1
Traffic accidents	3
TOTAL	22

[a] Cited from a report by Urage 1974 (ref. 2).

[b] Autopsied cases.

MORTALITY OF PATIENTS WITH YUSHO

Omae reported in 1975 that 29 deaths occurred among 1,291 patients with Yusho up to April 30, 1975 (ref. 1). Causes of these deaths were not given, however. Urabe also reported on 22 deaths seen among 1,200 patients until September 13, 1973, and referred to their causes (ref. 2). As shown in table 13, 9 of 22 deaths were caused by malignant neoplasms, suggesting a possible excess of deaths from cancer. Since some essential information needed for epidemiological analysis is not available to us, no further reference can be made with certainty to such a possibility at the present time.

CONCLUSION

As mentioned earlier, we demonstrated the presence of PCDF's in "Yusho oil" at a much higher concentration than expected. We also showed that PCDF's are relatively more concentrated in liver. Although neither the chemical nature nor the toxicity of PCDF's contained in "Yusho oil" and in the bodies of patients are known yet, our findings clearly indicate the necessity to pay greater attention to PCDF's for clarification of the nature of Yusho. Furthermore, our studies suggested the possible formation of PCDF's from PCB's when used as heat transfer medium. Beside this, another possibility that PCDF's might be formed by heating PCB's with peroxides, which are well known to be formed during heating cooking oils, should also be investigated.

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DISCUSSION

MR. ALLEN GREY (IIT Research Institute, Chicago, Illinois): Do you have any feel whether dibenzofurans concentrating in the body are more rapid in metabolism than the result of polychlorinated biphenyls?

DR. KURATSUNE: Unfortunately I have no definite idea. It is a more potent compound than PCB, I guess. I really cannot say.

DR. ORVILLE PAYNTER (EPA, Washington, D.C): Are there any reproductive problems or irregularities continuing for many years after the ingestion of these materials?

DR. KURATSUNE: There is some disturbance of menstruation. There have been some disturbances and there have been some related problems.

MS. DEBORAH A. BARSOTTI (University of Wisconsin, Madison, Wisconsin): Were there are doings that suspected any widespread gastic ulcerations or an erosion in the patients—gastric ulcers?

DR. KURATSUNE: No, I do not think so. Some of the patients had very persistent disorders of the intestines, disorders of the digestive system, but I do not know if they were suffering as a result of this or not.

VOICE: Any residual changes in the sebaceous gland during the autopsy?

DR. KURATSUNE: No, I do not know very much about it.

VOICE: I would like to ask one more question. Have you been able to specifically identify any of the dibenzofurans other than those in the body?

DR. KURATSUNE: You are asking if we could identify any of the dibenzofurans? No.

PATHOLOGICAL FINDINGS ASSOCIATED WITH CHRONIC EXPERIMENTAL EXPOSURE TO PCB's

Renate D. Kimbrough, M.D.*

Abstract

The principle organs affected by long-term exposure to commercial mixtures of polychlorinated biphenyls (PCB's) in rodents, mink, and monkeys are the gastrointestinal tract, the liver, and the lymphatic system. Lesions of the gastric mucosa have been described in monkeys, and ulcerations of the gastric mucosa have been observed in the rat. Pathological changes in the liver in many species, such as the monkey, rodent, and mink, consist of hepatomegaly, lipid accumulation, and liver cell necrosis, particularly at higher dietary levels. Hyperchromatic nuclei and mitotic figures are noted. Ultrastructural changes in the cytoplasm of affected hepatocytes consist of an increase in smooth endoplasmic reticulum, atypical mytochondria, and accumulation of concentrically arranged membranes, which usually surround lipid vacuoles. Particularly in rodents, accumulation of a brown pigment has been observed in Kupffer cells and macrophages (ceroid pigment and uroporphyrin).

In the mouse, (male Balb/cJ strain) neoplastic nodules (hepatomas, hyperplastic nodules) developed after dietary exposure to Aroclor 1254. Female Sherman strain rats fed 100 ppm Aroclor 1260 for 21 months developed hepatocellular carcinomas as well as neoplastic nodules (hyperplastic nodules) of the livers.

In the rat and the mouse, adenofibrosis of the liver occurred after exposing rodents to either Aroclor 1254 or Aroclor 1260. Adenofibrosis, a persistent lesion, does not disappear upon cessation of exposure to the PCB's.

In connection with the persistence of the different lesions mentioned in the rodents as well as in primates, it was noted that certain homologues of the PCB mixtures fed to the animals were not eliminated from adipose tissue and liver in any appreciable amount. In rats, 6 months feeding of Aroclor 1254 at a dietary level of 100 ppm followed by a 16-month recovery period resulted in total adipose tissue levels of 152 ppm penta, hexa-, and heptachlorobiphenyl. These animals still showed pathological changes in the liver, and liver PCB levels of 4.5 ppm.

INTRODUCTION

For the past 45 years, polychlorinated biphenyls (PCB's) have been used in transformers, capacitors, as heat transfer fluids, and have had other industrial applications (ref. 1). The toxicity of these and related compounds has recently been reviewed (ref. 2). A number of new observations have been made since a conference was held on PCB's in 1971 and it is the purpose of this paper to outline additional findings that are associated with chronic experimental exposure of animals to PCB's.

To study the long-term toxicity of the PCB's, commercial mixtures have usually been employed. This was done for two reasons: similar mixtures were found in the environment, and the individual constituents of the PCB's are very expensive and not readily available in the quantity necessary to conduct long-term studies. Table 1 gives the composition of some of the commercial PCB mixtures. If the PCB's are given as a single dose, they are of a low order of toxicity (table 2). The acute dermal toxicity is also low. It is the repeated and often long-term exposure to these compounds that may lead to a cumulative toxic effect.

HEPATOTOXICITY

Dietary exposure of rats to the PCB's produces enlargement of the liver cells in some adult rats at a dietary level of 5 ppm (0.4 mg/kg/day) (ref. 3). Liver weights were increased in 21-day-old F_1 male weanlings when the dams were fed as little as 1 ppm of Aroclor 1254 (ref. 3). At a dietary level of 20 ppm or above of Aroclor 1254 or 1260, which is equivalent to a dietary intake of 1.5 mg/kg/body weight per day, for an extended period of time, the hepatocytes in many rats had foamy cytoplasm consistent with lipid accumulation. In addition, hyperchromatic nuclei and binucleation was observed in many liver cells. Inclusions were present in the cytoplasm of the liver cells. These inclusions on electron microscopic examination were consistent with concentrically arranged membranes surrounding lipid vacuoles. Other ultrastructural changes observed at this and higher dietary levels in rats consisted of an increase in smooth endoplasmic reticulum and atypical mytochondria. These ultrastructural changes have also been described in primates (ref. 4) and are known to occur with a number of other xenobiotics. Similar observations were made in a study with smaller numbers of animals

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Table 1. Typical percentage composition of polychlorinated biphenyl products[a]

Homologue # Cl/biphenyl	Aroclor 1221	Aroclor 1016	Aroclor 1242	Aroclor 1254
0	11	<0.1	<0.1	<0.1
1	51	1	1	<0.1
2	32	20	16	<0.5
3	4	57	49	1
4	2	21	25	21
5	<0.5	1	8	48
6	ND[b]	<0.1	1	23
7	ND	ND	<0.1	6

[a] Percent (w/w) by GC/mass using area correction factors by homolog response, (Papageorge, written communication, June 1973).

[b] None detected; <0.01% = ND.

Table 2. The incidence and type of liver lesions in female sherman strain rats

	Incidence	
Type of Lesion	Controls	Experimental
Hepatocellular carcinoma	1/173	26/184
Neoplastic nodules	0/173	144/184
Foci or areas of cytoplasmic alteration	28/173	182/184

where the rats were fed Aroclor 1242 and Aroclor 1016 (ref. 5).

A brown pigment representing ceroid pigment and uroporphyrin was observed in Kupffer cells and macrophages. Particularly at higher dietary levels, livers showed pink fluorescence under UV lights.

In addition, exposure of rats and mice to PCB's for 6 months or longer produced grayish white lesions in the liver. On microscopic examination, these lesions consisted of proliferation of glandular epithelial cells that formed ducts and were surrounded by very pronounced fibrosis (refs. 6,7). The ducts often contained cellular debris. This lesion, called adenofibrosis, was first described by Edwards and White (ref. 6) in rats fed the carcinogen butter yellow (P-dimethylaminoazobenzene).

When rats were exposed for 6 months to a dietary intake of 500 ppm Aroclor 1254 and then sacrificed first at monthly and then at bimonthly intervals, the adenofibrosis persisted through a 10-month observation period. Usually adenofibrosis in rodents occurs concomitantly with hepatocellular carcinomas and/or neoplastic nodules (hyperplastic nodules). This was true for the Balb/cJ mouse (ref. 7). When 50 Balb/cJ inbred male mice were fed 300 ppm Aroclor 1254 (49.8 mg/kg/bodyweight per day), a total of 10 neoplastic nodules (hyperplastic nodules) were found in 9 per 22 surviving mice after 11 months of exposure to the experimental diet. No tumors were found in 58 surviving controls.

In another study, 400 weanling 21- to 26-day-old Sherman strain COBS female rats were distributed into two groups of 200 animals according to a table of random numbers. The animals were housed 10 rats per cage. Two hundred rats were fed plain ground purina chow and 200 rats were fed the same diet containing 100 ppm Aroclor 1260. This study was only conducted at this dietary level and with female rats because in a preliminary feeding study, a bladder tumor was found in 1 of 10 female rats fed 100 ppm Aroclor 1260 (ref. 8). The food consumption of the rats and their bodyweights were determined at intervals. A slight decline in the rate of the weight gain of the test group compared with the control group began about 3 months after onset of the experiment. Mean final bodyweights were 420 grams for the control group and 392 grams in the test group. This difference was statistically significant ($P < 0.001$) since such a large group of animals was involved in this study. The food consumption of rats declines with age. Accordingly, PCB intake declined from 11.6 mg/kg/day during the first week of exposure to 6.1 mg/kg/day at 3 months of exposure and to 4.3 mg/kg/day at 20 months. We determined the PCB concentrations in the experimental diet at intervals and these ranged usually from 70 to 107 ppm. We also spot checked the diet for aflotoxins. Aflotoxins were never detected in either the control or the experimental diet. A total of 173 control and 184 experimental animals survived to the age of 21-23 months. At autopsy of these rats, a consistent difference in the appearance of the livers was observed between the experimental and the control groups. Almost all (170 of 184 livers) of the experimental animals had from a few to multiple elevated tan nodules on the liver surface. Additional nodules were usually seen on sectioning. These nodules varied from .1 to several centimeters in diameter. In contrast, the liver of only one control animal showed gross abnormalities and was markedly enlarged, nodular, tan, and firm. A variety of tumors of other organs was observed in both the experimental and the control group but a difference in the incidence of tumors in other organs in the experimental and the control group did not exist.*

Histologic examination of the liver showed that 26 experimental rats and 1 control rat had hepatocellular carcinomas. An additional 144 experimental animals had hepatocellular nodules consistent with neoplastic nodules (hyperplastic nodules). In addition, 182 treated and 28 control animals had areas of hepatocytes with altered cytoplasm. The cells in these areas were often similar to those that were observed in the neoplastic nodules but the architecture of these atypical areas in the liver was the same as that of normal liver tissue and the liver plates merged with the surrounding liver tissue rather than compressing it.

In classifying the different liver lesions, the recommendations made in a liver tumor workshop (ref. 9) were followed.

The results of this bioassay test illustrate that Aroclor 1260 produces hepatocellular carcinomas according to traditional histological criteria and neoplastic nodules, which are a part of the spectrum of a response to hepatocarcinogenic agents. Neoplastic nodules must be included in the evaluation of tumorigenesis. Neoplastic lesions of the liver have also been produced by some of the commercial Japanese PCB mixtures; in mice with Kanechlor 500, and in Donryu rats with Kanechlor 400 (refs. 10,11).

The effect on the gastric mucosa as it has been described in primates (ref. 4) does not seem to be as prevalent in rodents. Only very high dietary levels, such as single doses of 3,000 mg/kg, will cause alteration of the gastric and duodenal mucosa (ref. 2). Primates (ref. 4) receiving 100 to 300 ppm Aroclor 1248 developed thickened gastric mucosa that contained numerous large cysts filled with mucin. Extension of the atypical appearing glandular epithelium into the muscularis mucosa and the underlying submucosa was also noted. This hyperplastic gastritis was quite persistent in the primate.

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*About 40 percent of the rats in each group had extrahepatic tumors. In a number of the rats, multiple tumors were present.

In conjunction with some of the feeding studies in rodents, tissue levels were determined in adipose tissue and the liver. It was noted that the gas chromatograms of the PCB's that were extracted from tissues was quite different from gas chromatograms of the standards. The PCB compounds remaining in adipose tissue as well as liver even after dietary exposure to PCB's has been terminated for as long as 10 or 16 months were the pents-, hexa-, and heptachlorobiphenyls, with molecular weights of 324, 358, and 392 (ref. 6). When rats were fed Aroclor 1254 at a dietary level of 100 ppm for 6 months, and were then removed from exposure to PCB's for 16 months, levels of 4.4 ppm of PCB-derived material was present in the liver and 152 ppm of PCB-derived material in adipose tissue on a wet weight basis. The livers of these rats showed some of the morphological changes already described (ref. 12). In primates that consumed Aroclor 1248 and reached adipose tissue levels of 127 ppm, after an 8-month recovery period the adipose tissue levels were 34 ppm of PCB-derived materials (ref. 4).

SUMMARY

The administration of PCB's to animals—particularly rodents but also monkeys and mink—produces a variety of responses in the liver. Early enlargement of the liver is primarily due to hypertrophy of the cells and an increase of the smooth endoplasmic reticulum of the cytoplasm of the liver cells. The smooth endoplasmic reticulum may condense in the liver cells and form hyalin inclusions, which on electron microscopic examination consist of concentrically arranged membranes containing lipid vacuoles. In addition to these lipid vacuoles, lipid accumulation may also occur throughout the liver cells. These changes are initially reversible. However, if the exposure to PCB's is continued, then liver damage, including liver cell necrosis, may also develop. This is accompanied by proliferation of the liver cells resulting in pleomorphism. Long-term exposure to PCB's in rodents produces a tumorigenic response which consists of the development of atypical areas, neoplastic nodules, and hepatocellular carcinoma. Adenofibrosis, which usually occurs concomitantly with hepatocellular carcinomas, has also been produced in the rat and the mouse. Pigment deposition within the macrophages and Kupffer cells has been noted (ref. 6). This pigment is composed of ceroid pigment (lipofuchsin) and uroporphyrin. Hemosiderin may also be present. In addition to liver changes, hyperplasia of the gastric mucosa has been observed in the primates (ref. 4) following the exposure to PCB's.

Because of these findings in experimental animals, ingestion of PCB's in humans must be curtailed. Exposure from all sources in the occupational environment needs to be reviewed and workers with long-term occupational exposure to PCB's should be studied. An effort should be made to establish all environmental sources of PCB's in order to assess exposure of the general population.

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DISCUSSION

MR. LAWRENCE ROY: You indicated after an animal has been exposed for 6 months you get regression, and if they did regress . . .

DR. KIMBROUGH: I don't know if I can answer all of that; you may have to repeat part of your question. But, first of all, I think in the studies that I did--and we really haven't got any time to go into all of this this morning--if you feed animals for only 6 months you do not see any tumors; you have to feed animals PCB's for much longer periods of time.

The first indication of any tumor development that I saw in reproduction studies we did was when the animals were exposed for at least 300 days. On the other hand, in the rat you do get what I call adenofibrosis if you feed the animals for about 6 months. That lesion is also known as cholangiofibrosis. Adenofibrosis does not regress. We did a feeding study where you feed the animals for 6 months, and then sacrifice them at bimonthly intervals, and the lesion will still be there 10 months later. So this is one problem with PCB's.

And at least some of the PCB homologs are retained for such a long period of time that I don't know whether you could also have what you might call internal exposure.

I think you had some other questions?

VOICE: First of all, do tumors cause death in these animals, and do these tumors cause an increase in size?

DR. KIMBROUGH: Some of the animals I think have died with large tumors at the end of the study. I don't think the tumors that are called hepatocellular carcinomas would regress. I also did not say that we had any regression in the animals we fed for 6 months.

MS. DIANE HORVAK (University of Wisconsin, Department of Pathology): What was the difference between the carcinoma and the nodule?

DR. KIMBROUGH: We did use histological criteria. In our classification, the tumors that we call neoplastic nodules were well circumscribed tumors that would extend over several lobules and some of them were actually quite large, and would compress the surrounding normal liver tissue, and because of that, they were circumscribed.

The lesions that were classified as carcinomas showed disorganized liver. Liver plates were two or three layers thick. You would have dilated sinusoids, would see mitotic figures and a great deal of pleomorphisms, but none of these tumors metastisized.

CHAIRMAN RALL: There's a question over there.

DR. ROBERT RISEBROUGH (University of California): Since we now know that these PCB preparations do contain dibenzofurans, I wonder if it is your plan to repeat these experiments with PCB's that do not contain the dibenzofurans. I wonder also if you would care to guess whether these tumors are caused by PCB or by the contaminants.

DR. KIMBROUGH: I have no plans at the moment to repeat these experiments. I do not know if I could get a completely clean Aroclor 1260.

The PCB that we used was Aroclor 1260 with the lot number AK3, which Dr. Bowes has analyzed; I think the level of the combined furans in that particular sample is less than 1 ppm. I may be wrong there, but if you recalculate that, you will find that the amount of furans consumed by the animals was very small. The amount of PCB's which the animals ingested was 5 mg/kg body weight/day, and the furans constituted less than I ppm of that. This amount is so small that I wonder whether it had any effect in this particular experiment.

CHAIRMAN RALLS: We'll come back to the issue of dibenzofurans later.

SUMMARY OF TOXICOLOGICAL STUDIES ON COMMERCIAL PCB's

J. C. Calandra, M.D., Ph.D.*

Abstract

A broad toxicological program was conducted to evaluate the biological effects of Aroclor 1242, 1254, and 1260 in mammalian as well as avian species. No-effect levels were defined which permit an assessment of the hazard these materials present to man and his environment.

The urgent need for additional toxicological data became apparent when the refinement of analytical techniques made possible the identification of PCB's in many portions of the ecosystem.

To assess the biological hazards of these materials, chronic feeding studies in rats and dogs were conducted according to accepted traditional procedures with Aroclors 1242, 1254, and 1260. It should be pointed out that Aroclor 1260 is no longer produced in this country since it does not meet the physical specifications for its restricted uses (ref. 1).

In addition, three-generation two-litter reproduction and teratology studies in albino rats as well as a dominant lethal mutagenic study in albino mice were considered to be necessary for the broad spectrum safety evaluation of these materials.

Effects such as decreases in eggshell thickness described in certain avian species suggested that toxicity and reproduction studies in chickens would be helpful in evaluating possible untoward effects in birds. A list of the studies conducted are presented in table 1.

The experimental design of the chronic rat and dog studies is given in tables 2 and 3. It should be noted that these studies were of typical Food and Drug Administration design for the evaluation of the safety of direct and indirect food additives.

The important results in the chronic rat studies which were conducted on Charles River rats are summarized briefly in tables 4, 5, and 6.

Food consumption, mortality, and hematologic, urine, and blood chemistry studies did not differ among treatment and control groups. The only weight depression observed was at the 24-month point in females fed 100 ppm of Aroclor 1254.

Liver weight increases were noted in males and females fed 100 ppm Aroclor 1254 or Aroclor 1260, and in females only in the 100 ppm group fed Aroclor 1242.

As expected, the important histopathologic changes in the rat study were present in the livers of the 24-month sacrifice animals and consisted of hepatocellular alterations such as focal hypertrophy, cytoplasmic lipid changes and in some animals at 100 ppm, hepatomas or cholangiohepatomas. No evidence of hepatocellular carcinogenicity of the Aroclors was found in this study.

The conclusion that "no evidence of hepatocellular carcinogenicity of the Aroclors was found in this study" was reached only after extensive reevaluation of the original liver slides as well as additional liver sections from all of the animals after the Kimbrough results on Aroclor 1260 became known to us (ref. 2).

The slides were read independently and separately by Dr. Donovan Gordon (ref. 3), Professor Ward Richter (ref. 4), and Professor P. Pour (ref. 5) of Industrial BIO-TEST Laboratories, the University of Chicago, and the Eppley Institute for Cancer Research, respectively.

The interpretation of pathologic changes that occur in the liver as a result of absorption of chlorinated hydrocarbons is a key issue that needs resolution by the community of pathologists (ref. 6, 7). The problem is highlighted in the context of PCB's since there appears to be disagreement as to whether Aroclor 1260 is or is not a hepatocellular carcinogen (ref. 8). Pour reevaluated the Kimbrough slides and does not agree with the reported findings.

The findings in the chronic dog study are summarized in table 7. The feeding of Aroclor 1260 at 100 ppm showed an increase in serum alkaline phosphatase activity and liver weights. A slight decrease in body weight gain was noted at the 100 ppm dose level in males and at 10 and 100 ppm in females. No remarkable histopathologic changes were found.

The design of the dominant lethal mutagenic study is presented in table 8. No effects related to Aroclor treatment were seen in the parameters listed in table 9—mortality, mating index, number of implantation and resorption sites, number of viable embryos, preimplantation loss or mutation rates. This finding is in agreement with that of Green et al. (ref. 10).

No effects related to Aroclor treatment were seen in the parameters listed in table 9—mortality, mating index, number of implantation and resorption sites, number of viable embryos, preimplantation loss or mutation rates. This finding is in agreement with that of Green et al. (ref. 10).

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The standard design for a three-generation two-litter reproduction study in albino rats is given in table 10 and the teratology study in table 11. A brief summary of the results (table 12) indicates that none of the Aroclors studied produced adverse effects in the two litters of the first generation.

All progeny delivered by female rats in each of three generations exposed to dietary levels of up to 100 ppm of either Aroclor 1242, 1254, or 1260 were structurally normal. A reduction in the mating index was observed in the second and third generations of animals fed either 10 or 100 ppm. The ability of females to conceive, carry the delivery process to parturition, and to successfully nourish the young was not affected by the three Aroclors. It should be stressed that no changes in the reproductive tract of either male or female rats were produced by any of the three Aroclors. Findings in the third generation were similar to those in the first with no suggestion of any alterations in response as a function of succeeding generations.

Teratologic studies in which albino rats were exposed to either Aroclor 1242, 1254, or 1260 at doses of up to 30 mg/kg during rapid organogenesis—gestation days 6 through 15—were conducted in our laboratories. No evidence of embryotoxicity as reflected by an increase of fetal resorption or teratogenicity as measured by complete external, skeletal, and internal evaluation of fetuses obtained from the Aroclor-treated females, was obtained. Other experimental data pertaining to the potential teratogenicity of polychlorinated biphenyls supports the lack of adverse effects. Mizunuya (ref. 11) and his colleagues found no evidence of teratogenicity in the rat when fed at dietary levels of up to 250 ppm. In the mouse, Toeruek (ref. 12) found that doses of up to 500 mg/kg were nonteratogenic although when given on gestation days 1 through 6 a decrease in implantation sites and fetal weights was observed. These doses given on gestation days 7 through 11 failed to produce any changes with respect to either reproductive parameters or teratogenicity. Further, no evidence of malformation was obtained in infant monkeys delivered to females fed either 2.5, 5, or 25 ppm Aroclor 1248 although the birth weights were reported to be reduced (ref. 9).

The last study in this series was a toxicity/reproduction study in white leghorn chickens (tables 13, 14, and 15).

Aroclor 1260 at all test levels did not produce adverse effects on the various parameters investigated. Egg production in hens fed 100 ppm Aroclor 1242 or 1254 was decreased as was egg hatchability. In fact, poor hatchability of eggs from hens fed 8 ppm Aroclor 1242 was found (table 15).

In addition, Aroclor 1242 at 10 and 100 ppm and Aroclor 1254 at 100 ppm were associated with reduced eggshell thickness. Chick viability was affected by both substances at the 10 ppm dose level.

CONCLUSIONS

1. The no-effect level for the Aroclors in the chronic rat and dog studies is about 10 ppm.
2. No teratogenic or mutagenic effects were found.
3. No hepatocellular carcinomas were present.
4. The no-effect level in the rat reproduction study is between 1 and 10 ppm and is the result of low mating indices.
5. In the chicken reproduction and teratology studies, effects were more severe with Aroclor 1242 with the no-effect level being 2 to 4 ppm.

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DISCUSSION

MR. PAUL AGENTINE: You didn't find any effects or pathological agents in the dog?

DR. CALANDRA: Would you repeat that?

MR. ARGENTINE: How did you arrive at the no-effects level in canine at 10 ppm? In studies you showed no pathological agents and no toxicity?

DR. CALANDRA: As I indicated, this is a very brief and rapid summary. We have a question about liver weights and one or two other parameters and to be conservative, we have used the number of 10 ppm. In other words, we are not in any way indicating that PCB's are nontoxic materials, and this is a conservative estimate.

DR. ALLEN GREY (I.I.T. Research, Chicago, Illinois): Are you able to relate the differences in biological effects on the various Aroclors to their composition?

DR. CALANDRA: Only generally. As everyone knows, the higher chlorinated materials appear to be more persistent in the mammalian system. And there appears to be a target organ—the liver—which appears to be more susceptible to the higher chlorinated materials. I think these are the only generalizations you can make at this time.

Table 1. Toxicity studies conducted with Aroclors 1242, 1254 and 1260

Type of test	Test animal
Two-year chronic oral	albino rats
Two-year chronic oral	beagle dogs
Three-generation reproduction	albino rats
Teratology	albino rats
Dominant lethal mutagenic	albino mice
Toxicity/reproduction	white leghorn chickens

Table 2. Two-year chronic oral toxicity study--albino rats and beagle dogs

Test material	Dietary levels (ppm)	Number of animals per dietary level			
		rats		dogs	
		M	F	M	F
None	- - -	50	50	4	4
Aroclor 1242	1, 10, 100	50	50	4	4
Aroclor 1254	1, 10, 100	50	50	4	4
Aroclor 1260	1, 10, 100	50	50	4	4

Table 3. Two-year chronic oral toxicity study--albino rats and beagle dogs

Parameters investigated:

- Body weight
- Food consumption
- Hematology
- Clinical blood chemistry (BUN, SAP, SGPT, fasting blood glucose, SGOT--dogs only)
- Urinalyses
- Pathology (gross, organ weights, microscopic)

Table 4. Ingestion of Aroclor 1242 by albino rats--liver effects

Increase in liver weights--100 ppm--females	
Primary liver lesions	
None at 3-, 6-, 12-month sacrifice	
24-month sacrifice--100 ppm--increased incidence of:	
Nodular hyperplasia	(8/20)
Hepatoma	(2/20)
Cholangiohepatoma	(1/20)
Hepatocellular carcinoma	(0/20)

Table 5. Ingestion of Aroclor 1254 by albino rats--liver effects

Increase in liver weights--100 ppm	
Primary liver lesions	
None at 3-, 6-, 12-month sacrifice	
24-month sacrifice--100 ppm--increased incidence of:	
Nodular hyperplasia	(13/27)
Hepatoma	(4/27)
Cholangiohepatoma	(2/27)
Hepatocellular carcinoma	(0/27)

Table 6. Ingestion of Aroclor 1260 by albino rats--liver effects

Increase in liver weights--100 ppm	
Primary liver lesions	
None at 3-, 6-, 12-month sacrifice	
24-month sacrifice--100 ppm--increased incidence of:	
Nodular hyperplasia	(7/27)
Hepatoma	(5/27)
Cholangiohepatoma	(2/27)
Hepatocellular carcinoma	(0/27)

Table 7. Effects of ingestion of Aroclors--beagle dogs

Test material	Effect
Aroclor 1242	no effects
Aroclor 1254	slight decrease in body weight gain --100 ppm
Aroclor 1260	slight decrease in body weight gain --100 ppm, males --10 and 100 ppm, females increase in SAP--100 ppm increase in liver weights--100 ppm

Table 8. Dominant lethal mutagenic study--albino mice

Test material	Dose*
Corn oil	0.9 ml/kg
Methyl methane sulfonate (MMS)	100 mg/kg
Aroclor 1242	500 or 1000 mg/kg
Aroclor 1254	500 or 1000 mg/kg
Aroclor 1260	500 or 1000 mg/kg

*Single dose given i.p. to 12 males/group.

Table 9. Dominant lethal mutagenic study--albino mice

No effects related to Aroclor treatment seen in:

- Mortality
- Mating index
- Number of implantation sites
- Number of resorption sites
- Number of viable embryos
- Preimplantation loss
- Mutation rates

Table 10. Three-generation reproduction study[a]--albino rats

Test material	Dietary levels (ppm)			Number of animals per dietary level	
				M	F
None	-	-	-	8	16
Aroclor 1242	1,	10,	100	8	16
Aroclor 1254	1,	10,	100	8	16
Aroclor 1260	1,	10,	100	8	16

[a] Two litters per generation.

Table 11. Teratology study--albino rats

Test material	Dose[a] (mg/kg/day)	Number of gravid rats
Corn oil	-	26
Aroclor 1242	10 or 30	26
Aroclor 1254	10 or 30	26
Aroclor 1260	10 or 30	26

[a] Administered on gestation days 6 through 15 (10 doses).

Table 12. Effect of Aroclors on reproduction/teratology--albino rats

1. Reproduction study
   First generation--no effects
   Second and third generations--10 and 100 ppm
   — decrease in mating index
   — decrease in incidence of pregancy
   (Aroclor 1242--no third generation with 100 ppm).
2. Teratology study--no effects.

Table 13. Toxicity/reproduction study--white leghorn chickens

Test material	Dietary levels (ppm)	Number of animals per dietary level	
		M	F
None	-	8	40
Aroclor 1242	1, 2, 4, 8, 10, 100	4	20
Aroclor 1254	1, 10, 100	4	20
Aroclor 1260	1, 10, 100	4	20

Table 14. Toxicity/reproduction study--white leghorn chickens

Parameters investigated:

- Body weight
- Food consumption
- Egg production
- Egg quality
- Egg hatchability
- Eggshell thickness
- Chick body weight
- Chick viability
- Pathology

Table 15. Effects of Aroclors on chicken reproduction

Parameter	Dietary level causing effect	
	1242	1254
Egg production	100	100
Egg hatchability	8	100
Shell thickness	100	100
Chick viability	10	10

Aroclor 1260--no effects at any dose level

PATHOBIOLOGICAL RESPONSES OF PRIMATES TO POLYCHLORINATED BIPHENYL EXPOSURE

J. R. Allen, D.V.M., Ph.D., and D. H. Norback*

Abstract

Male and female rhesus monkeys received varying levels of polychlorinated biphenyls (PCB's) and were evaluated for toxic effects, reproductive dysfunctions, and metabolism of the compounds. Female rhesus monkeys exposed to dietary levels as low as 2.5 and 5.0 ppm of PCB (Aroclor 1248) developed facial acne, erythema, subcutaneous edema, conjunctivitis, and loss of eyelashes. Reproductive dysfunctions were manifested by irregular menstrual cycles, early abortions, and stillbirths. As a result of transplacental migration of the compounds, all infants born of PCB-exposed animals contained PCB's in their tissues at birth. The infants, which continued to be exposed to PCB's by ingestion of milk from their lactating mothers, developed skin lesions and 50 percent expired within 4 months.

Metabolic studies demonstrated 90 percent absorption of the PCB's from the gastrointestinal tract and distribution in organs of high lipid content. Hydroxylated metabolites were formed in the liver and excreted through the biliary and urinary routes. Lower chlorinated congeners were more rapidly metabolized and excreted, while concentrations of the highly chlorinated biphenyls persisted in the adipose tissue in excess of 2-1/2 years. The detection of the urinary metabolite trans-3,4-dihydro-3,4-dihydroxy-tetrachlorobiphenyl suggests that the mechanism of metabolism is through an arene oxide intermediate. In vivo and in vitro studies demonstrated binding of PCB's with macromolecules.

INTRODUCTION

Even though the polychlorinated biphenyls (PCB's) have been used extensively for various industrial purposes for the past 40 years, the health significance of human exposure to these compounds only recently has become of widespread concern. The scientific community was alerted to the potential environmental health problem by Jensen in 1966 (ref. 1) after PCB's were identified in tissue extracts of birds experiencing reproductive difficulties in Sweden. The magnitude of the problem was brought to the forefront by the "Yusho" incident when over 1,000 Japanese suffered prolonged ill effects from exposure to PCB-contaminated rice oil (ref. 2). Further concern about the potential danger of PCB's on human health followed disclosure of increasing levels of these compounds in various foods. The contamination has been attributed to incorporation of these compounds within the food chain or from packaging of food products in PCB-impregnated paper containers (ref. 3). Increasing levels of PCB's in human tissue samples attest to the magnitude of the human exposure (refs. 4,5).

Within this laboratory various animal models, including the nonhuman primate and rodent, have been evaluated following exposure to PCB's for the development of lesions which parallel those recorded in man. Emphasis has been placed on determining pathophysiological alterations that arise in animal models as a result of exposure to several levels of PCB's for variable periods of time. The absorption, tissue distribution, and rate of excretion of PCB's have been determined. The interaction of the PCB's or their metabolites with cellular macromolecules has also been a major area of investigation. The following report, which includes previously unpublished observations, presents a summary of the progress that has been made in this laboratory on the above mentioned areas of PCB research.

GENERAL EFFECTS OF PCB'S ON NONHUMAN PRIMATES

The investigation of toxicity produced by PCB's in various animal species demonstrated the limitations of rodents as animal models. Male Sprague-Dawley rats were able to survive for 1 year on diets containing 100 ppm PCB (Aroclor 1248, 1254, or 1262) without showing signs of illness (ref. 6). These observations substantiated those of Keplinger et al. (ref. 7). Increasing the PCB content of the rat diets to 1,000 ppm did not produce skin lesions; however, death occurred within 6 to 8 weeks due to widespread hepatic degeneration (ref. 8).

Male rhesus monkeys developed many of the signs experienced by humans that had been inadvertently exposed to PCB's. Monkeys fed diets containing 100 and 300 ppm PCB (Aroclor 1248) (table 1) developed facial edema, erythema, acne, and alopecia within 3 weeks. The lesions became progressively more severe with increased length of exposure (refs. 9,10). In addition, the animals developed anorexia, loss in weight, hypoproteinemia, hypolipidemia, and anemia. Within 3 months the majority of the monkeys had died or were moribund. Necropsies of these animals revealed decided mucosal gastric hyperplasia with penetration of the glandular epithelium into the underlying submucosa (ref. 10). Numerous ulcerations of the hyperplastic gastric mucosa were also present (ref. 11). In addition, there was a decided hypertrophy of the liver.

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Female monkeys fed 25 ppm PCB (Aroclor 1248) (table 1) in the diet developed facial lesions similar to those observed in the animals receiving higher levels of PCB's (ref. 12). After 2 months on the PCB diet it was necessary to discontinue the exposure due to the severity of intoxication. One of the six experimental animals died 4 months after the initial exposure to PCB's. Necropsy evaluation demonstrated severe gastric hyperplasia and ulceration. The surviving adult female monkeys continued to be devoid of eyelashes and displayed facial acneform lesions 2 years following exposure to the PCB's. Infants born to these females were small (350 vs. 450 g) and contained PCB's in their tissues at birth.

Female monkeys given 2.5 and 5.0 ppm PCB (Aroclor 1248) (table 2) in their diets developed facial edema, swollen eyelids, erythema, loss of hair, and acne within 2 months (ref. 13). By the fourth month, irregularities in the menstrual cycles and an increased level of urinary ketosteroids were recorded (ref. 14). Following 6 months of PCB exposure the female monkeys were bred to control males. Six of eight animals on the 5.0 ppm diet conceived (table 2). The remaining two were bred on five separate occasions without conceiving. Four of the six females experienced abortion early in gestation. Eight of eight of the 2.5 ppm PCB fed animals conceived; however, only five were able to carry their infants to term. As was the case with infants of animals given the higher levels of PCB's, all the infants were small and at birth their skin contained detectable levels of PCB's.

The infants were permitted to nurse their mothers for 4 months. Within 2 months focal areas of hyperpigmentation, swollen lips and eyelids, loss of eyelashes, and acneform lesions of the face developed. The skin of these infants showed a decided increase in the PCB level over this period. Within 4 months, 3 of the 6 infants died due to PCB intoxication. After weaning, the remaining three have shown improvement of the skin lesions during the 4-month period.

Four adult male rhesus monkeys were also exposed to a diet containing 5.0 ppm PCB's (Aroclor 1248) (table 1) for 17 months (average total intake of PCB's 460 mg). They began to develop a slight periorbital edema after 6 months of exposure; however, it was much less severe than in the female monkeys receiving a similar level of PCB. The morphological features and viability of the spermatozoa as well as the ability to fertilize control female rhesus monkeys was unaffected during the initial 12 months of PCB exposure. Subsequently one of the four males lost weight and developed alopecia, acne, periorbital edema and decreased libido. A testicular biopsy of this animal showed a decided hypoactivity of the seminiferous tubules. There was an absence of mature spermatozoa and a predominance of Sertoli cells of the tubules. The remaining three males have remained healthy and sexually active (ref. 15).

ABSORPTION, METABOLISM, TISSUE DEPOSITION, AND EXCRETION OF PCB's

Over 90 percent of a single oral dose (1.5 or 3.0 g per kg) of PCB's (Aroclor 1248) given to adult rhesus monkeys was absorbed from the gastrointestinal tract. Chromatographic analysis of the tissues 14 days after exposure revealed a predominance of higher chlorine isomers that had a predilection for the adipose tissue and organs containing a high fat content (ref. 16).

Rhesus monkeys fed 25 ppm PCB (Aroclor 1248) in the diet attained levels of 127 μg/g within the adipose tissue after 2 months. Eight months after discontinuation of exposure to PCB's the levels were 34 μg/g within the adipose tissue. After 33 months, the levels within the adipose tissue ranged from 3 to 14 μg/g; the residues contained greatly increased proportions of highly chlorinated congeners. Transplacental movement of the PCB's was demonstrated by the presence of PCB's in the infants born to exposed females. The tissues of an infant, born to a female 8 months following the discontinuation of PCB's in the diet, contained 25 μg/g of PCB in the fat and adrenal tissues at the time of birth. In an infant born to a female 29 months following the discontinuation of PCB's, the levels within the adipose tissue were 3.38 μg/g at 4 months of age.

Female monkeys given 5.0 ppm PCB in their diets attained maximum levels of PCB's within their adipose tissue at 6 months (141 to 177 μg/g adipose tissue). However, it required approximately 14 months on the 2.5 ppm diet for the monkeys to reach similar maximum PCB levels in their adipose tissue (126 to 144 μg/g). Males which received 5.0 ppm PCB's attained levels

Table 1. Experiments on exposure of primates to PCB's (Aroclor 1248*)

Level of PCB in diet (ppm)	No. of animals	Length of exposure (months)	Total PCB intake
300	6 males	3	3.6 to 5.4 g
100	6 males	2	0.8 to 1.0 g
25	6 females	2	250 to 400 mg
2.5	8 females	16-19	243 to 303 mg
5.0	8 females	16-19	460 to 614 mg
5.0	4 males	17	530 to 692 mg

*Monsanto Co., Inc., St. Louis, Missouri.

Table 2. Modification in reproduction in primates that were exposed to Aroclor 1248 in the diet

	Control	2.5 ppm	5.0 ppm
Total impregnated (no./no. animals)	12/12	8/8	6/8
Resorptions or abortions (no./no. animals)	0/12	3/8	4/8
Stillborn (no./no. animals)	0/12	0/8	1/8
Normal births (no./no. animals)	12/12	5/8	1/8

ranging from 128 to 200 μg per g adipose tissue at 14 months. Infants born of mothers exposed to 2.5 and 5.0 ppm PCB's within the diet contained concentrations of PCB's ranging from 1.0 to 4.8 μg/g within the skin at birth. While nursing from mothers consuming PCB diets, the infants continued to accumulate the compound. At 3 months, the levels within the tissues ranged from 86 to 136 μg/g. The concentration of PCB's within the milk ranged from 0.15 to 0.40 μg/g. The tissues of the infants which died while nursing PCB-fed mothers contained high levels of PCB's within the thymus, ovaries, brain, kidneys, adrenal glands and pancreas (20-48 μg/g tissue). Lower levels were found in the liver, lymph nodes, and bone marrow (8-16 μg/g).

Monkeys and rats demonstrate species variation in the metabolic response to the PCB congener 2,5,2',5'-tetrachlorobiphenyl (TCB). Over 66 percent of the single dose (500 mg/kg) administered to rats was recovered from the feces, and an additional 10 percent was present in the urine within the initial 72 hours (ref. 17). The material present in the body was concentrated within the adipose tissue. There was a transient high level of TCB within the blood at 24 hours. Other organs which contained significant quantities of TCB, however at lower concentrations, included the liver, skin, and muscle. The major urinary metabolite was identified as 3-OH-2,5,2',5'-tetrachlorobiphenyl. Other monohydroxy TCB metabolites were present in minor quantities (ref. 17).

Over 90 percent of an oral dose (500 mg/kg) of ^{3}H TCB administered to infant rhesus monkeys was absorbed from the gastrointestinal tract and was highly concentrated in the skin, adrenal gland, liver, and adipose tissue. At 72 hours, less than 2 percent of the dose had been eliminated in the urine and 1 percent in the feces. Monohydroxy TCB, a major metabolite present in the rat urine, was a minor metabolite in the urine of the monkeys. The two major metabolites were dihydroxy-TCB and trans-3,4-dihydro-3,4-dihydroxy TCB (ref. 18). A second minor metabolite was hydroxy-3,4-dihydro-3,4-dihydroxy TCB.

Following the oral dose of ^{3}H-TCB (1 g/kg) to juvenile monkeys, a major percentage of the material was absorbed from the gastrointestinal tract and was highly concentrated in the adrenal, adipose tissue, and skin. Significant quantities were present within the liver, muscle, and uterus. After 4 weeks, 14.8 percent of the dose was secreted into the bile. Approximately 75 percent of the biliary material was reabsorbed by the gut and the nonabsorbed material was recovered from the feces during this period. Over 90 percent of the PCB's excreted in the bile was in the form of water soluble glucuronic acid conjugates. An additional 8 percent of the total dose was recovered from the urine.

Administration of the higher chlorine congener ^{3}H 2,4,5,2',4',5'-hexachlorobiphenyl (HCB) to rats or monkeys demonstrated low levels of exretion into the bile. An oral dose of HCB (1 g/kg) administered to rats resulted in excretion of 0.3-0.7 percent of the dose per day in the bile over a period of 14 days. The urine was free of detectable levels of radioactivity (ref. 19). At 14 days, 65 percent was recovered from the body tissues. The compound was highly concentrated within the adrenal glands, adipose tissue, and skin and in the female within the ovaries and uterus. Following the administration of a single dose of HCB (1 g/kg) to juvenile rhesus monkeys, less than 2 percent was excreted via the biliary-fecal route over a 3-week period. There was no detectable radioactivity within the urine. The organs with the highest concentration of the material included the adrenal, adipose tissue, and skin. Due to the large mass of muscular tissue, this was a major reservoir of the compound.

INTERACTION OF METABOLITES WITH CELLULAR MACROMOLECULES

Following the administration of ^{3}H 2,5,2',5'-tetrachlorobiphenyl (TCB) to infant rhesus monkeys, interaction of TCB and macromolecules of cells and of serum was evaluated (ref. 20). Separation of the serum constituents by polyacrylamide gel electrophoresis demonstrated association of the TCB primarily with serum albumin. Over 90 percent of the radioactivity of liver homogenates eluted from a Sephadex G-25 column was in the protein and nucleic acid fractions. The majority of the macromolecular-associated HCB apparently was bound by hydrophobic association. Extraction of liver homogenates with hexane, precipitation of the hexane-extracted homogenate with TCA, and subsequent extraction of the TCA precipitate with methanol resulted in extraction of the majority of the radioactivity. In the extracted residue, 1.1 percent of the radioactivity remained which may represent covalently bonded material. Recent in vitro studies employing monkey microsomes incubated with an NADPH generating system demonstrated 20 percent of the metabolized ^{3}H-TCB was bound to microsomal protein and RNA in a nonextractable form. Binding of ^{3}H-TCB was prevented by heating the microsomes to 100°C prior to incubation (ref. 21).

DISCUSSION

These experiments employing nonhuman primates have demonstrated development of parallel signs and lesions of PCB intoxication in humans and rhesus monkeys exposed to similar levels over comparable periods of time. Acne, subcutaneous edema of the face, and edema of the eyelids were observed in man (ref. 22) and lower primates (ref. 15) exposed to PCB's. The facial signs of PCB exposure appear to be a sensitive indicator of PCB intoxication. The rhesus monkeys after exposure for 2 months to dietary levels of PCB's (2.5 and 5.0 ppm) developed facial alterations after a total consumption of 32-50 mg of PCB's. It is noteworthy that PCB levels of 5.0 ppm are presently permitted in foods destined for human consumption.

The most debilitating lesions in the monkeys were the severe hyperplasia and ulceration of the stomach. Whether similar changes occur in the stomach of man exposed to PCB's remains to be clarified. Nausea and anorexia described by the human subjects suggest potential gastric alterations. Liver hypertrophy, proliferation of the endoplasmic reticulum, and increased hepatic microsomal enzyme activities were observed in man (ref. 23) and in lower primates (ref. 11).

Menstrual irregularities, decreased libido, occurrence of stillborns, reduced birth weights, and transplacental movement of PCB's in humans and rhesus monkeys have been recorded (refs. 13,24). The reproductive failures of monkeys exposed to PCB's were due to inability to maintain a pregnant state. The majority of the animals did not experience appreciable difficulty in conception; however, a large percentage of the animals aborted during the first 45 days of pregnancy. These observations suggest an inability of implantation or inability to maintain the implanted embryo during the early stages of pregnancy.

Although the mechanism of reproductive dysfunction has not been clarified, there is some indication of hormonal modifications. Alterations in the urinary ketosteroids were reported in humans exposed to PCB's (ref. 22). Increased levels of urinary ketosteroids have been observed in the nonhuman primates that experienced reproductive failures (ref. 14). One mechanism of altered steroid metabolism may be secondary to the increase in the hepatic mixed function oxidases that are present in the hypertrophic livers of exposed animals. It has also been a consistent observation that the organs associated with steroid production, the adrenals and ovaries (particularly the corpora lutea), have contained relatively high concentrations of PCB's in exposed animals. Thus the compounds may possibly have a direct effect on these organs.

The presence of PCB's in the milk of other species has been previously reported (ref. 3). This avenue of infant exposure and the potential morbidity and mortality was vividly demonstrated in the infant monkeys who nursed from mothers exposed to 2.5 and 5.0 ppm in the maternal diets. The presence of relatively low levels of PCB's in the diets of lactating females represents a potential source of PCB intoxication to nursing infants.

Metabolic studies of the rhesus monkey demonstrate over 90 percent absorption of the PCB's from the gastrointestinal tract following oral administration. The material is concentrated in organs with high lipid content, including the adipose tissue, skin, adrenal, corpora lutea of the ovaries, and brain. Within the liver the greatest portion of the material is associated with the membranes of the endoplasmic reticulum.

Studies with the single congener TCB demonstrate the metabolism of the compound to hydroxylated forms which are conjugated with glucuronic acid and excreted into the bile. Enterohepatic circulation of the PCB's undoubtedly occurs as only 20 percent of the material secreted into the bile was recovered from the feces. The greatest portion of the metabolized TCB was excreted through the urinary system. The more highly chlorinated biphenyl HCB was more slowly eliminated from the body via the biliary-fecal route; HCB or metabolites were not detected within the urine. The facilitated metabolism and excretion of the lower chlorine congeners was also indicated by the relative decreased storage of these compounds, and conversely the accumulation of higher chlorinated congeners, within the adipose tissue.

Metabolic studies that have been conducted on nonhuman primates suggest mechanisms of interaction of PCB's with tissues and, more importantly, indicate potential mutagenic and carcinogenic effects of the PCB's. Metabolites have been isolated from rhesus monkeys exposed to the PCB congener 2,5,2′,5′-tetrachlorobiphenyl that are formed through an arene oxide intermediate (ref. 18). Similar metabolites of the PCB's and the potential for arene oxide formation has been demonstrated in rabbits (refs. 25,26). Arene oxides formed by the metabolism of other aromatic hydrocarbons have been shown to covalently bind with macromolecules and produce mutagenic and carcinogenic changes in mammalian cells (ref. 27). Dechlorination of the more highly chlorinated biphenyls, demonstrated by dechlorination of 2,4,5,2′,4′,5′-hexachlorobiphenyl (ref. 28), provides a mechanism through which metabolism of highly chlorinated biphenyls through an arene oxide intermediate would be facilitated.

Evidence demonstrating the association of PCB's with liver macromolecules supports the theoretical potential of the PCB's for covalent binding with cellular macromolecules. Thus, it appears that alkylation of macromolecules is one mechanism by which the PCB metabolites cause widespread injurious effects. Further credence for the ability of the compound to produce alterations in the macromolecules is presented in recent reports of hepatocellular tumors developing in rats and mice exposed to PCB's (refs. 29-31).

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DISCUSSION

VOICE: I'm from the Massachusetts Society. Have you examined the samples for minute contaminants?

DR. ALLEN: That is a good question. I presume your primary interests are in the dibenzofurans. The Monsanto Company has volunteered to analyze the Aroclor 1248 used in our experiments for the furans. In our discussion last week they were hopeful of having these data available for this conference. If Dr. Wright is in the audience perhaps he would give us a progress report on the subject. (No answer from the audience.) I can say that we have done some preliminary work in this area and have found undetectable levels of furans in the samples. However, more detailed studies to clarify this question are underway at the present time.

VOICE: How can you determine that the PCB's are covalently bound to macromolecules?

DR. ALLEN: Repeated extractions carefully monitored for radioactivity are the best methods of removing any absorbed material from the protein. Standard gel chromatography methods do not differentiate between adsorption and covalently bound materials. We hope to be able to generate enough PCB bound to macromolecules to permit the determination of the exact covalent nature of the bond following macromolecular digestion.

VOICE: Which macromolecule did you use?

DR. ALLEN: We were using protein and RNA from monkey microsomes. These microsomes were incubated with ^{3}H PCB in a NADPH generating system. The protein and RNA were isolated subsequently and their radioactivity determined.

VOICE: Could you tell us roughly how much PCB your animals consumed per kilogram of body weight?

DR. ALLEN: The female animals on the PCB experiments weighed between 6 and 7 kilograms. Table 1 gives the average total intake of PCB's by these animals during the various experiments.

PCB CHLORINATION VERSUS PCB DISTRIBUTION AND EXCRETION

H. B. Matthews, Ph.D.* and M. Anderson, Ph.D.†

Abstract

The distribution and excretion of selected ^{14}C-labeled polychlorinated biphenyls (PCB's) were studied in the male rat. The distribution and excretion of each of the PCB's was studied after either i.v. or oral administration. Following administration, each of the PCB's was rapidly removed from the blood and stored in the liver and muscle. The rates of PCB redistribution from liver and muscle to skin and adipose tissue and/or elimination in urine was related to the degree of chlorination. The subsequent rates of PCB removal from skin and adipose tissue, excretion in urine, and total excretion were related to the degree and position of chlorination of the biphenyl molecule. None of the PCB's studied was excreted to a significant extent prior to metabolism to more polar compounds. It appears as if the degree and position of chlorination controls the rate of metabolism. The limiting factor in metabolism may be the facility of arene oxide formation as a metabolic intermediate. The toxicologic implications of arene oxide formation versus PCB accumulation in animal tissues are discussed.

INTRODUCTION

Due to the complexity of commercial polychlorinated biphenyl (PCB) formulations and the difficulty experienced by others in their efforts to interpret the results of toxicological studies in which these mixtures were used, we have chosen to study selected individual PCB's. We believe that detailed studies of individual PCB's, chosen to represent industrial PCB formulations, may not only afford an insight into the biological fate of the more complex commercial formulations, but may also be used in the construction of pharmacokinetic models of the distribution and excretion of these and other chlorinated hydrocarbons. Our ultimate goal is to have pharmacokinetic models which will permit the accurate extrapolation of animal data to man.

Four of the PCB's studied were 4 chloro-, 4,4'-dichloro-, 2,4,5,2',5'-pentachloro-, and 2,4,5,2',4',5'-hexachlorobiphenyl. These PCB's have degrees of chlorination similar to, and are constituents of Aroclors 1221, 1232, 1254, and 1260, respectively. Each of the PCB's studied was labeled with carbon -14.

METHODS

All of the data presented in this paper were obtained with an i.v. dose of 0.6 mg PCB/kg body weight; however, studies with the pentachlorobiphenyl at doses ranging from 0.06 to 6.0 mg/kg failed to show any effect of dose on the distribution and excretion of this PCB (ref. 1). The results presented in this paper were obtained by i.v. injection of the PCB's; however, these results have been repeated without different results by studies in which the PCB's were administered by oral intubation (ref. 2). In these studies, the time points of sampling ranged from 15 minutes to 7 days for each of the PCB's and up to 42 days for the penta- and hexachlorobiphenyls. Three animals were treated and sacrificed at each time point and the data presented represent the average values obtained. The total radioactivity in the tissue samples was determined by oxidation and liquid scintillation counting.

RESULTS

Following i.v. injection, approximately 90 percent of the total dose of each PCB was removed from the blood within 15 minutes. Initially, most of the administered dose was stored in the liver and muscle. Within 15 minutes after administration, the liver contained 15 to 30 percent of the total dose of each of the PCB's (figure 1). Removal of mono- and dichlorobiphenyl from liver was primarily via excretion in the bile in the form of several metabolites; only trace amounts of the parent compounds were excreted in the bile. The pentachlorobiphenyl was removed from the liver by metabolism and excretion in the bile and by redistribution to other tissues, whereas the primary mechanism for the removal of hexachlorobiphenyl from the liver was redistribution. Due to the relatively large mass of muscle, total storage of the PCB's in muscle was similar to that observed in liver (figure 2). It is assumed that the only mechanism of PCB removal from muscle was by redistribution to tissues having a higher affinity for these compounds (ref. 2).

Most of the long-term storage of PCB's in the body was in the skin and adipose tissue. Since one of the earliest symptoms of chronic intoxication by PCB's and certain other chlorinated hydrocarbons is chloracne, a skin disorder (ref. 3), data on the accumulation of these

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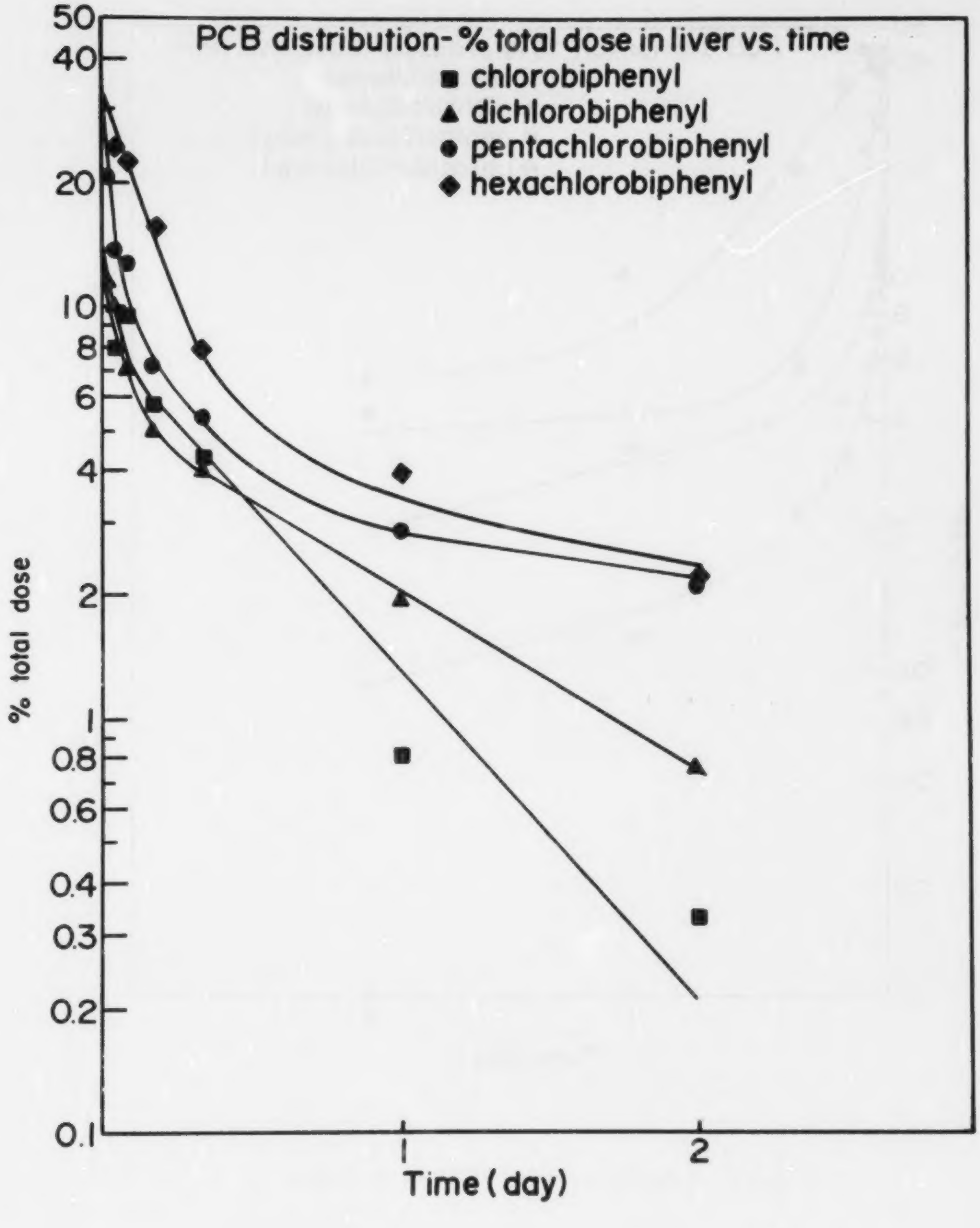

Figure 1. Rate of removal of PCB's from liver.

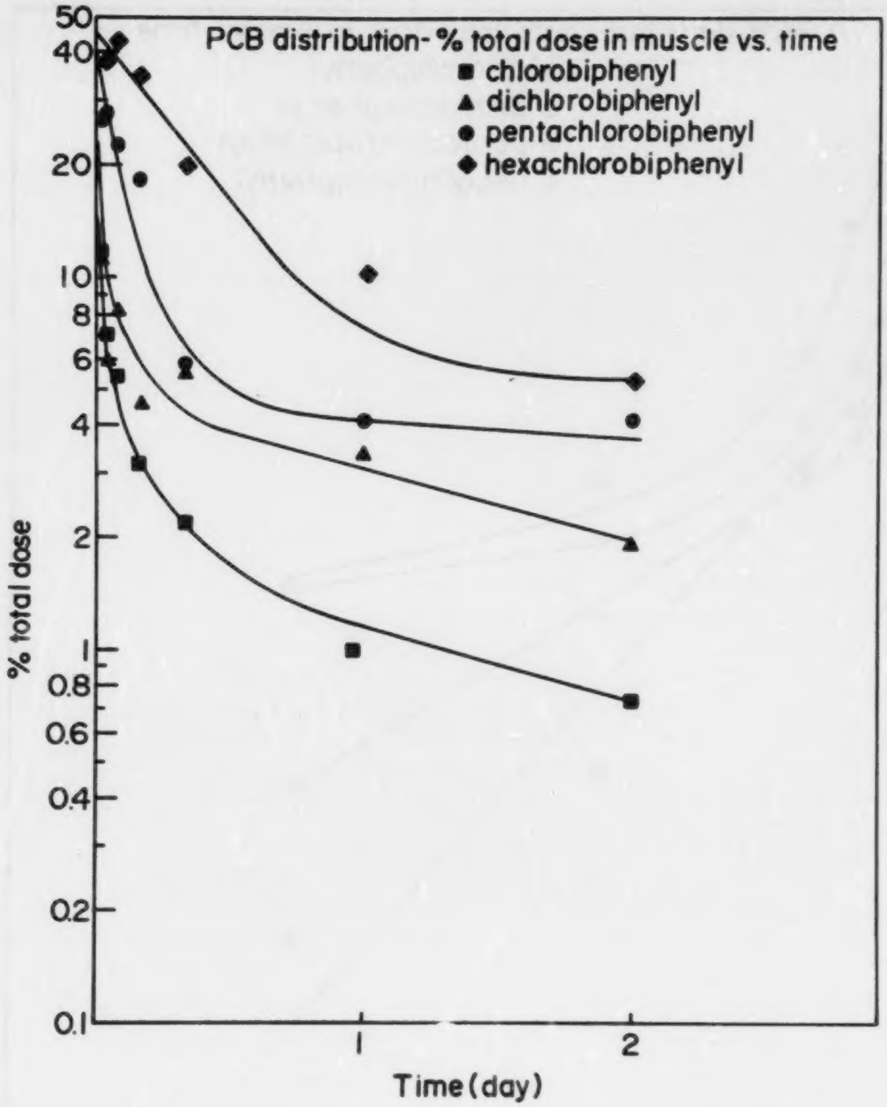

Figure 2. Rate of removal of PCB's from muscle.

compounds in skin was of particular interest (figure 3). It may not be obvious in figure 3, due to the long time scale of 8 hours to 42 days, but the uptake of PCB's by skin was slower than the uptake by liver and muscle (ref. 2). The rate of PCB removal from skin decreased as the degree of chlorination of the PCB's increased, and following the removal of approximately 25 percent of the hexachlorobiphenyl, little further decay from skin was observed during the remainder of the 42-day study.

The accumulation of PCB's in adipose tissue was a slower process than that observed in skin (figure 4). The peak concentrations of mono-, di-, penta-, and hexachlorobiphenyl in adipose tissue were reached at 1 hour, 2 hours, 4 hours, and 7 days after administration, respectively. The magnitude of the peak concentrations in fat also tended to increase with increasing chlorination, whereas the rates of PCB removal from adipose tissue decreased with increasing chlorination of the PCB. Hexachlorobiphenyl concentrations in fat never showed a decline from peak concentrations.

The rates of PCB removal from tissues would be expected to be reflected in their rates of excretion. All rats kept for 1 day or longer were held in individual metabolism cages and fed food and water *ad libitum*. Both urine and feces were collected daily. The most dramatic effect of increasing chlorination of the biphenyl molecule was seen in the percent of the total dose excreted in the urine (figure 5). Cumulative excretion in urine for 7 days accounted for approximately 60, 34, 8, and less than 1 percent of the total mono-, di-, penta-, and hexachlorobiphenyl dose, respectively Greater than 90 percent of the material excreted in the urine was in the form of one or more conjugated metabolites of the given PCB (ref. 2).

Cumulative excretion of the PCB's in feces was a more consistent process which, with the exception of the hexachlorobiphenyl, did not appear to be greatly affected by degree of chlorination (figure 6). Animals treated with penta- or hexachlorobiphenyl were held for up to 42 days. Extrapolation of the daily rates of total PCB excretion showed that, with the exception of hexachlorobiphenyl, excretion would eventually account for approximately 100 percent of the administered dose of each PCB. The excretion of hexachlorobiphenyl was so slow that extrapolation to infinite time indicated that less than 20 percent of the administered dose would ever be excreted.

DISCUSSION

Work in our laboratory and elsewhere indicates that only about 10 percent of each of these PCB's is excreted as the parent compound. We also know from extraction and analysis of the radioactivity in the tissues that appreciable amounts of the metabolites are not stored in the tissues (ref. 2). We have thus assumed that metabolism is a prerequisite to the excretion of PCB's. We have also shown that the rates of excretion and the rates of removal from skin and adipose tissues of mono-, di-, and pentachlorobiphenyl are inversely proportional to their degrees of chlorination (ref. 2). On the other hand, the very slow excretion of hexachlorobiphenyl would not have been predicted by this relationship. Therefore, an additional factor appeared to be affecting the rate of metabolism of hexachlorobiphenyl.

An examination of the molecular structures of these four PCB's shows that there is only one obvious difference, other than chlorination, among the structures of the hexachlorobiphenyl and the three other PCB's. The difference is that this hexachlorobiphenyl does not have two adjacent unsubstituted carbon atoms.

It was demonstrated in Williams' laboratory in the 1950's that chlorinated benzens that had two adjacent unsubstituted carbon atoms were metabolized and excreted 3 to 20 times more rapidly than benzenes with similar degrees of chlorination that did not have adjacent unsubstituted carbon atoms (refs. 4,5). Schulte and Acker suggested that a similar substitution pattern is required for the metabolism of PCB's (ref. 6). This suggestion was partially confirmed by Jensen and Sundstrom (ref. 7) when they showed that PCB's which did not have two adjacent unsubstituted carbon atoms were found in the highest concentrations in the tissues of higher animals and man. The hexachlorobiphenyl used in our studies was found in the highest concentrations of any PCB in human tissues. Our data on the distribution and excretion of this PCB and the fact this particular PCB is one of the more common constituents of the more highly chlorinated commercial PCB formulations explain why such high concentrations were found in human and animal tissues.

As a point of reference, we have also studied the distribution and excretion of similar doses of several chlorinated pesticides in the rat. The initial half-life of dieldrin was quite similar to that of pentachlorobiphenyl. The initial half-life of DDT was five- to six-fold longer than that of pentachlorobiphenyl and only Mirex and hexachlorobenzene, of the pesticides tested, had infinitely long half-lives such as that observed for hexachlorobiphenyl (ref. 8).

Two adjacent unsubstituted carbon atoms are important to the metabolism of the PCB's because their presence facilitates the formation of arene oxides. Arene oxides are formed by the hepatic mixed-function oxidases as intermediates in the metabolism of a number of lipophilic compounds, and these oxides have been

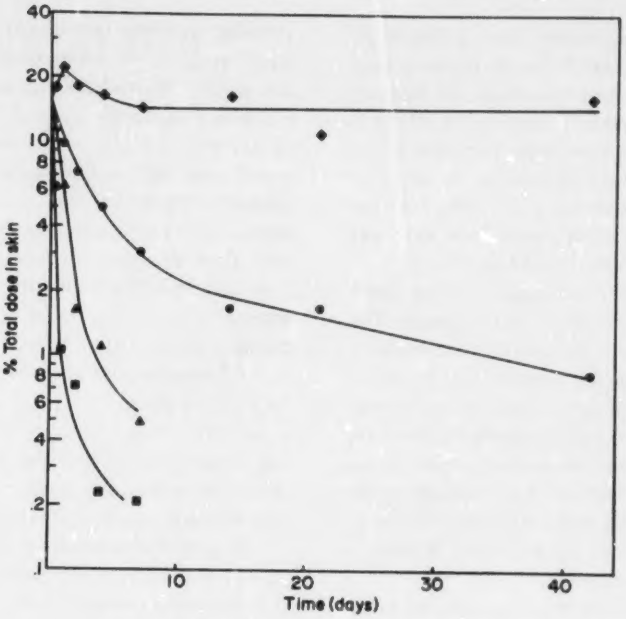

Figure 3. Rate of removal of PCB's from skin.

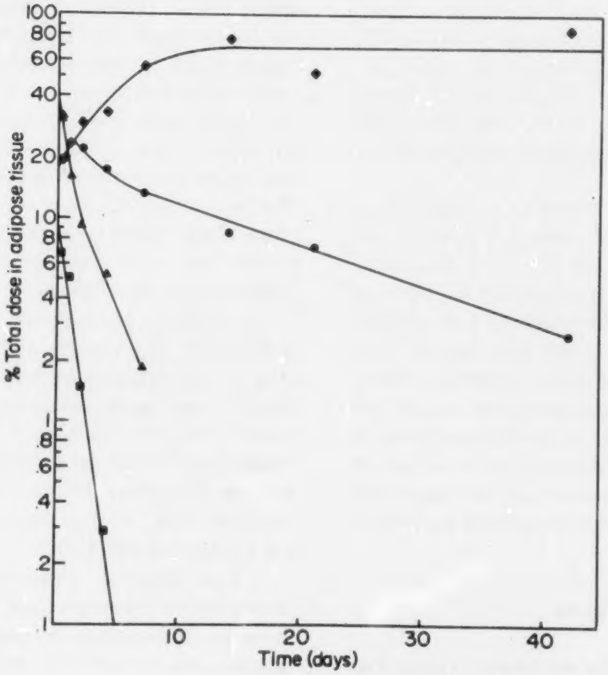

Figure 4. Rate of removal of PCB's from adipose tissue.

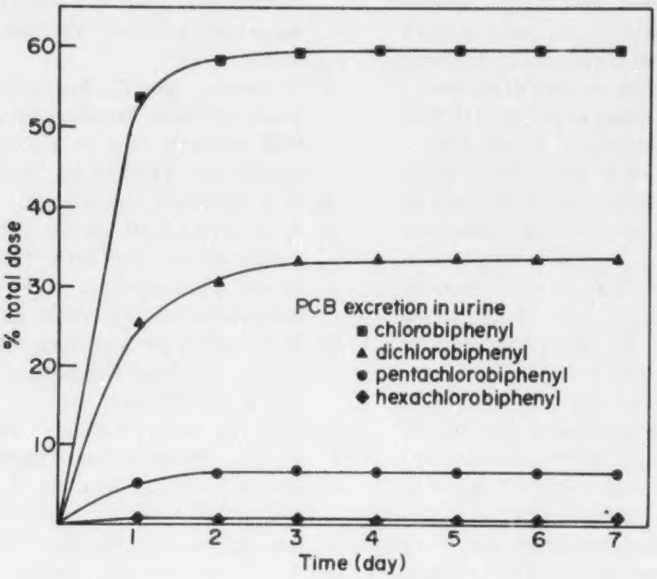

Figure 5. Cumulative excretion of PCB's in urine.

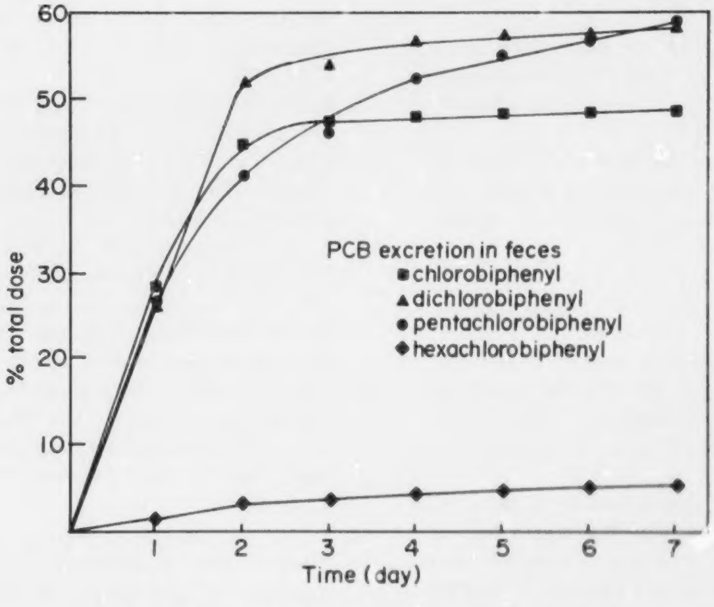

Figure 6. Cumulative excretion of PCB's in feces.

implicated as potential carcinogens (refs. 9,10). Evidence for arene oxides as intermediates in the metabolism of implicated as potential carcinogens (refs. 9,10). Evidence for arene oxides as intermediates in the metabolism of PCB's has been provided by Gardner et al. (ref. 11), Safe et al. (ref. 12), and in our own laboratory (ref. 13).

Thus, we are given a dilemma. Those PCB's which can be metabolized and excreted may be metabolized via a carcinogenic intermediate and those PCB's which are not readily metabolized have an extremely long biological half-life. Several researchers have provided evidence that the PCB's may be carcinogenic (refs. 14-16). On the other hand, Vos et al. (ref. 17) have shown the very slowly metabolized hexachlorobiphenyl used in this study to be acnegenic, to cause liver damage, and to induce hepatic porphyria. It is not yet known if it is the parent PCB's or their metabolites which account for the primate reproductive failures described by Allen (ref. 18). The only way that we are going to establish which of the PCB's or their metabolites are carcinogenic or are going to cause any of the other toxicological problems and at what levels of exposure the problems are likely to arise is through systematic pharmacokinetic studies, which will allow us to extrapolate the results of chronic low-dose environmental exposures from laboratory animals to man. Until such data are available, it is my opinion that every effort should be made to avoid environmental contamination by any type of PCB's.

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ENZYMATIC AND OTHER BIOCHEMICAL RESPONSES TO SELECTED PCB's

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Abstract

Isomerically-pure mono-, di-, tri-, tetra-, hexa-, and octa-chlorobiphenyls were injected i.p. into weanling male rats at a dosage of 50 mg/kg/day for 3 consecutive days, the animals being killed 96 hr after the last injection. The influence of position and degree of chlorination of the biphenyl nucleus on hepatic function was compared to that produced by purified biphenyl. Hepatic function was assessed by pentobarbital sleeping times and in vitro assays of p-nitroanisole O-demethylase, aniline hydroxylase, aminopyrine N-demethylase, carboxylesterase and sulfobromophthalein-glutathione conjugating enzyme activities. For the mono-oxygenases closely associated with the hepatic endoplasmic reticulum, enhanced induction of activity was observed with highly chlorinated biphenyls and by low chlorine-containing congeners having chlorine atoms substituted at the 4- and 4′-positions irrespective of chlorination at other positions. For those enzymes less discretely localized in the hepatocyte, the position of the chlorine atoms appeared to be less important. Interrelationships with other hepatic functions and the rate of chlorobiphenyl biotransformation is discussed.

Toxicologic assessment of commercial chlorobiphenyl mixtures has been complicated by the heterogeneity of the congeners, by marked differences in physical and chemical properties which, undoubtedly, influence rates of absorption, distribution, metabolism and excretion and by the possible presence of toxic impurities and byproducts (refs. 1-4). As techniques of definitive analysis have developed, the complexity of commercial chlorobiphenyls (Aroclors, Monsanto Industrial Chemicals, St. Louis, Mo.) has been revealed showing that, with the exception of Aroclor 1016 and 1232, one predominant congener composed of a number of positional isomers is found in each preparation (fig. 1). As the percentage of chlorine increases, the predominant congener shifts from a mono- to a tri- to a tetra- to a penta-chlorobiphenyl (refs. 5,6).

If it was possible to separate these complex mixtures, one could examine several facets of the toxicology of these compounds, and several questions could be posed and, perhaps, answered. Do the various congeners possess the same toxicological properties; i.e., do dichlorobiphenyls have the same effect as hexachlorobiphenyls? Do all of the tetrachlorobiphenyl isomers produce the same toxicologic responses? Are the pathologic and toxicologic alterations observed due to the biphenyl nucleus itself, to the positions occupied by individual chlorine atoms, or to the number of chlorines present on the biphenyl nucleus. Since hepatic enzyme induction has been a well-characterized phenomenon of adaptation to these chemicals, we attempted to answer some of the above questions using changes in hepatic ultrastructure and in enzyme activity as indices of structure-activity relationships.

Our first study, completed in 1972, and published in 1974, used a small series of isomerically-pure chlorobiphenyls of known position and degree of chlorination, synthesized and purified by my colleagues, Dr. O. Hutzinger and Dr. S. Safe (refs. 3,7). The objective of these experiments was to elicit responses (induction of hepatic drug-metabolizing enzymes) which, hopefully, could be related to the structures of the pure chlorobiphenyls. We injected young male Wistar strain rats intraperitoneally with 50 mg/kg of the agent, dissolved in peanut oil, for 3 consecutive days, the animals being killed 96 hr after the last injection. The livers were quickly removed and samples were taken and stained for light and electron microscopy. The remaining hepatic tissue was used for the preparation of microsomes and soluble supernatant for the enzymatic assays. These assays included representative functions of the microsomal mono-oxygenases (p-nitroanisole O-demethylase, aniline hydroxylase, aminopyrine N-demethylase), hydrolases (nonspecific carboxylesterase) and the conjugation of sulfobromophthalein (BSP) with reduced glutathione (GSH). Some of the results of that study are shown in the next few figures.

Figure 2 presents the results observed for the cytoplasmic enzyme system involved in conjugating BSP following exposure to DDT, Aroclor 1254 and 1260, biphenyl, and a series of pure chlorobiphenyls. Significant ($p<0.05$) increases in activity were observed with all agents tested. It should be noted that biphenyl caused a marked increase in this enzyme activity.

Figure 3 shows the influence of the DDT isomers, commercial Aroclors, biphenyl, and the series of isomerically pure chlorobiphenyls on pentobarbital-induced sleeping time. Highly significant ($p<0.05$) reductions in sleeping time were observed with the commercial Aroclors and DDT isomers. Biphenyl and 4-chlorobiphenyl caused no significant ($p > 0.05$) changes in sleeping time. Of the dichloro-isomers, only the 4,4'-isomer significantly reduced the duration of effect. Both tetrachloro-isomers caused significant reductions in sleeping time though the 2,5,2',5'-isomer was less effective. The hexa- and octa-chlorobiphenyls caused a marked reduction in sleeping times, being comparable to those observed with p,p'-DDT and the commercial Aroclors.

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Figure 4 summarizes the influence of the various agents tested on the hepatic mixed function oxidases aniline hydroxylase (A), p-nitroanisole O-demethylase (B), and aminopyrine N-demethylase (C). As has been observed by others, we found that the most responsive enzymes were those closely associated with the smooth endoplasmic reticulum. The results in vitro reflected the observations of altered sleeping times in vivo. One can see that, even with this limited number of pure chlorobiphenyls, not all caused effects of the same magnitude. To summarize the results of this study, treatment with biphenyl caused slight induction while 4-chlorobiphenyl did not. The mixed function oxidases were markedly induced by pure hexa- and octa-chlorobiphenyls and also by di- and tetra-chlorobiphenyls with chlorines substituted at the 4-positions of the rings. Considering the dichloro-isomers, when the 4- and 4'-positions were occupied, there was a much greater inductive effect for all of the enzyme activities than was observed with the 2,2'- and 2,4'-isomers. The same positional phenomenon was observed for the two tetra-chlorobiphenyls studied, the induction caused by 2,4,2',4'-tetra-chlorobiphenyl being much greater than that observed with the 2,5,2',5'-isomer. The results obtained for the higher chlorinated analogs suggested that the positions of the chlorine atoms were not as important. Our results, with the exception of the marked effects obtained following treatment with 4,4'-dichlorobiphenyl, confirmed the observations of other investigators who have found that penta-, hexa- and octa-chlorobiphenyls had greater enzyme-inducing potential than did low chlorine-containing biphenyls (refs. 8-11).

On the basis of our initial studies, we came to the conclusion that the biphenyl nucleus could exert some effect on hepatic enzyme levels (fig. 4) though the only function markedly affected was the BSP-GSH conjugation (fig. 2). Much more important conclusions were that not all congeners possessed the same toxicologic properties and that, while greater inductive effects were observed with highly chlorinated biphenyls, marked ultrastructural and enzymatic changes were observed with specific di- and tetra-chlorobiphenyls, particularly those with chlorines substituted on the 4-position on the ring. We have extended the investigation to a broader series of mono-, di-, tri-, and tetra-chlorobiphenyls in an attempt to confirm the importance of position of the chlorine on the ring structure of low chlorine-containing congeners. Isomerically-pure chlorobiphenyls, synthesized by my colleagues or purchased from Analabs Inc. (North Haven, Conn.), were injected intraperitoneally using the same regimen (50 mg/kg/day for 3 consecutive days, killing the animals 96 hr after the third injection). A 12,000 g-20 min supernatant from 20% w/v homogenates of liver was used as the enzyme source. This study has been published recently (ref. 12). Some of the pertinent results are shown in the next figures.

It was essential, before studying the effects of chlorination and position, to determine what effects the biphenyl nucleus had on hepatic enzymes. Table 1 shows the results of the i.p. administration of vehicle (peanut oil), commercially available biphenyl (Eastman Organic Chemicals, Rochester, N.Y.), and purified biphenyl on the activities of hepatic O-demethylase (OD), aniline hydroxylase (AH), carboxylesterase (CE), and the BSP-GSH conjugating enzyme. Treatment with unpurified biphenyl resulted in significant ($p < 0.05$) increases in activities of three of the enzymes. In contrast, biphenyl repurified by thin layer chromatography caused a significant increase only in BSP-GSH conjugating enzyme activity, suggesting the presence of an impurity in the commercial material. Since relatively pure chlorobiphenyls were to be used, repurified biphenyl was used for the control groups of animals.

The influence of monochlorobiphenyls on hepatic O-demethylase, aniline hydroxylase, carboxylesterase, and BSP-GSH conjugating enzyme activities are compared in table 2 with the effects following treatment with biphenyl. While changes in microsomal O-demethylase were not observed, all three monochloro-isomers significantly increased aniline hydroxylase and carboxylesterase levels. The BSP-GSH conjugating enzyme activity was not affected.

The influence of a series of di-, tri-, and tetrachlorobiphenyls on the selected enzymatic functions are shown in figures 5, 6, and 7, respectively. With each series of isomers, a chlorine atom on the 4-position caused a more marked induction of hepatic drug-metabolizing enzyme activities than did a chlorine atom at any other position. As one increased the degree of chlorination, subsequent substitution at the 2-position was next in importance followed by substitution at the 3-position. The results conclusively demonstrated that not all isomers of the mono-, di-, tri-, and tetrachloro-

Table 1. The effects of intraperitoneally administered peanut oil, commercial biphenyl, and purified biphenyl dissolved in peanut oil on enzyme activities of rat liver[a]

	Activities (in total wt of fresh liver/100 g body wt)[b]		
Enzyme	Vehicle	Commercial biphenyl	Purified biphenyl
p-Nitroanisole O-demethylase	168.0±23.9	211.5±38.7	195.6±19.1
Aniline hydroxylase	102.7±26.7	352.8±102.8[c]	97.3±15.1
Carboxylesterase	234.4±53.5	336.7±55.3[c]	236.2±24.7
BSP-GSH conjugating enzyme	5.0± 0.9	14.4± 1.9[c]	8.8± 0.9[c]

[a] The animals received 50 mg of biphenyl/kg (0.15-0.25 ml of solution) for 3 consecutive days and were killed 96 hr after the last injection. Vehicle-treated animals received peanut oil on the same volume basis. Twelve animals were treated with vehicle, 11 with commercial biphenyl, and 18 with purified biphenyl.

[b] The activities of O-demethylase and aniline hydroxylase are expressed as nanomoles of product formed/minute. Carboxylesterase activity is expressed as micromoles of substrate hydrolyzed/minute while the BSP-GSH conjugating enzyme activity is expressed as micrograms of conjugate formed/minute. Activities are presented in terms of the total liver/100 g of body wt.

[c] Values are statistically different from vehicle-treated control values at $p<0.05$.

Table 2. Effects of acute intraperitoneal administration of biphenyl and monochlorobiphenyls on hepatic enzyme activity

Treatment	n	Activities (in total wt of fresh liver/100 g body wt)[a]			
		OD[c] (nmol/min)	AH (nmol/min)	CE (μmol/min)	BSP (μg/min)
Biphenyl	18	195.6±19.1	97.3±15.7	236.2±24.7	8.8±0.9
2-Chlorobiphenyl	6	184.8±17.2	188.3±30.4[b]	309.2±35.1[b]	7.5±1.5
3-Chlorobiphenyl	6	193.6±28.1	225.8±55.3[b]	381.7±64.3[b]	9.8±1.5
4-Chlorobiphenyl	6	233.4±32.5	168.1±37.4[b]	306.2±24.5[b]	9.8±1.6

[a] Values presented are the mean ± SD of the number of animals per group.

[b] Values are significantly different from values obtained from biphenyl-treated animals, $p<0.05$.

[c] The enzymes investigated include p-nitroanisole O-demethylase (OD), aniline hydroxylase (AH), carboxylesterase (CE), and sulfobromophthalein-glutathione conjugating enzyme (BSP).

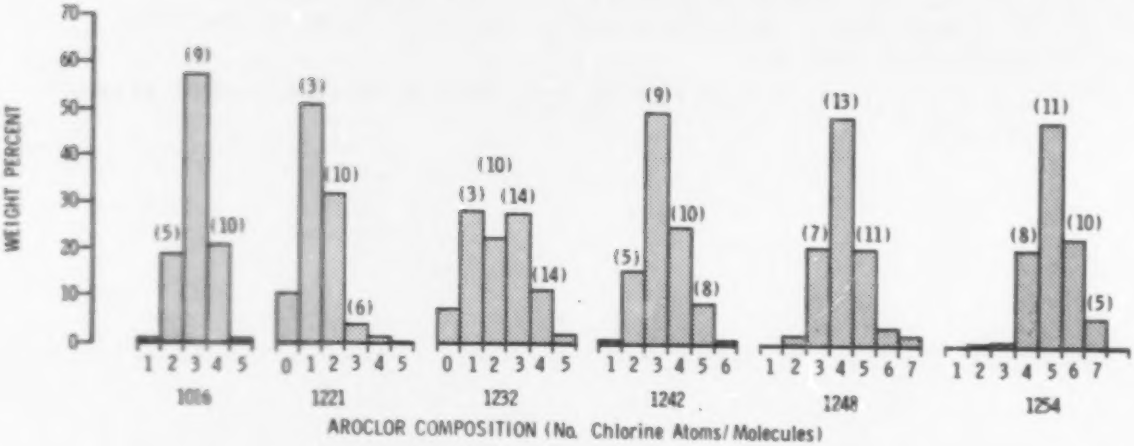

Figure 1. The congener composition of commercially available Aroclors based on the weight percent of biphenyls bearing different numbers of chlorine atoms/molecule. Data were obtained from reports by Webb and McCall (ref. 6), Sissons and Welti (ref. 5), and from information supplied by the Monsanto Industrial Chemicals Company.

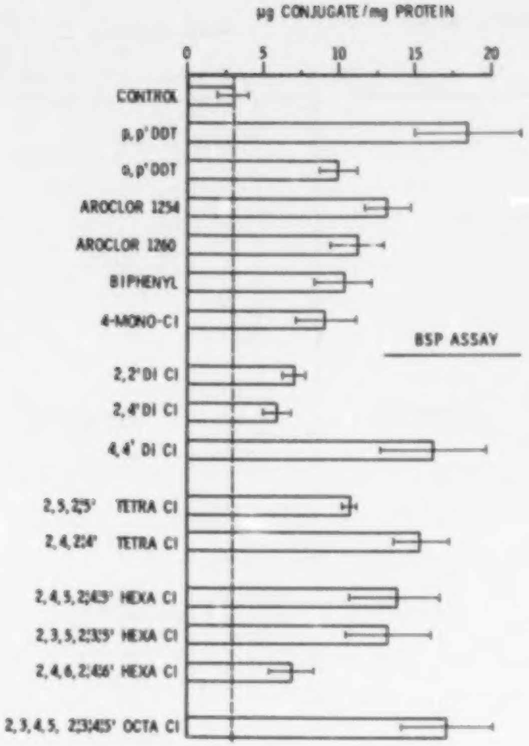

Figure 2. The effect of pretreatment of young male rats with DDT (o,p', and p,p'-isomers), Aroclors 1254 and 1260, biphenyl, and a series of isomerically pure chlorobiphenyls on the hepatic cytoplasmic enzyme which conjugates sulfobromophthalein (BSP) with reduced gluthathione. Activities are expressed as μg of conjugate formed mg^{-1} protein min^{-1}. Animals were treated by i.p. injection for 3 consecutive days, enzyme activity being determined 96 hr after the last injection. The values (bars) represent mean activities ±S.D. of the means of 19 control animals and 7 animals per treated group.

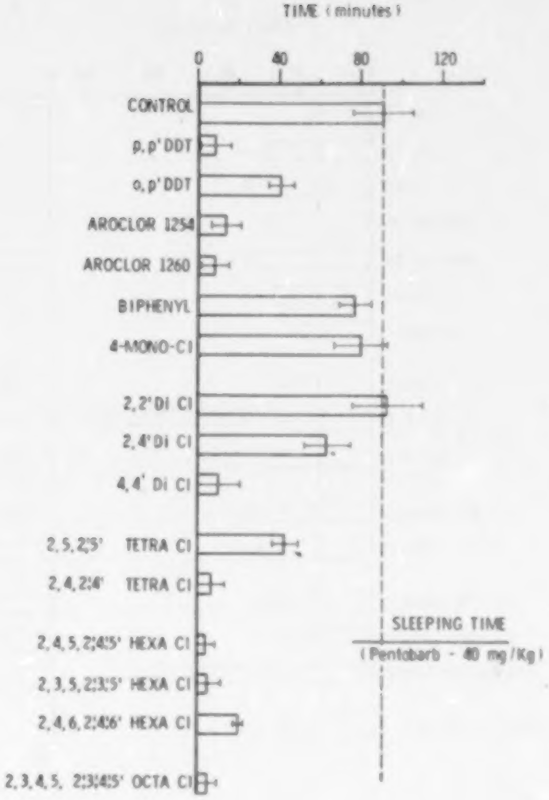

Figure 3. The effect of pretreatment of young male rats with DDT (o,p' and p,p'-isomers), Aroclor 1254 and 1260, biphenyl, and a series of isomerically pure chlorobiphenyls on the sleeping time produced by an injection of 40 mg/kg sodium pentobarbital. For other details, see figure 2.

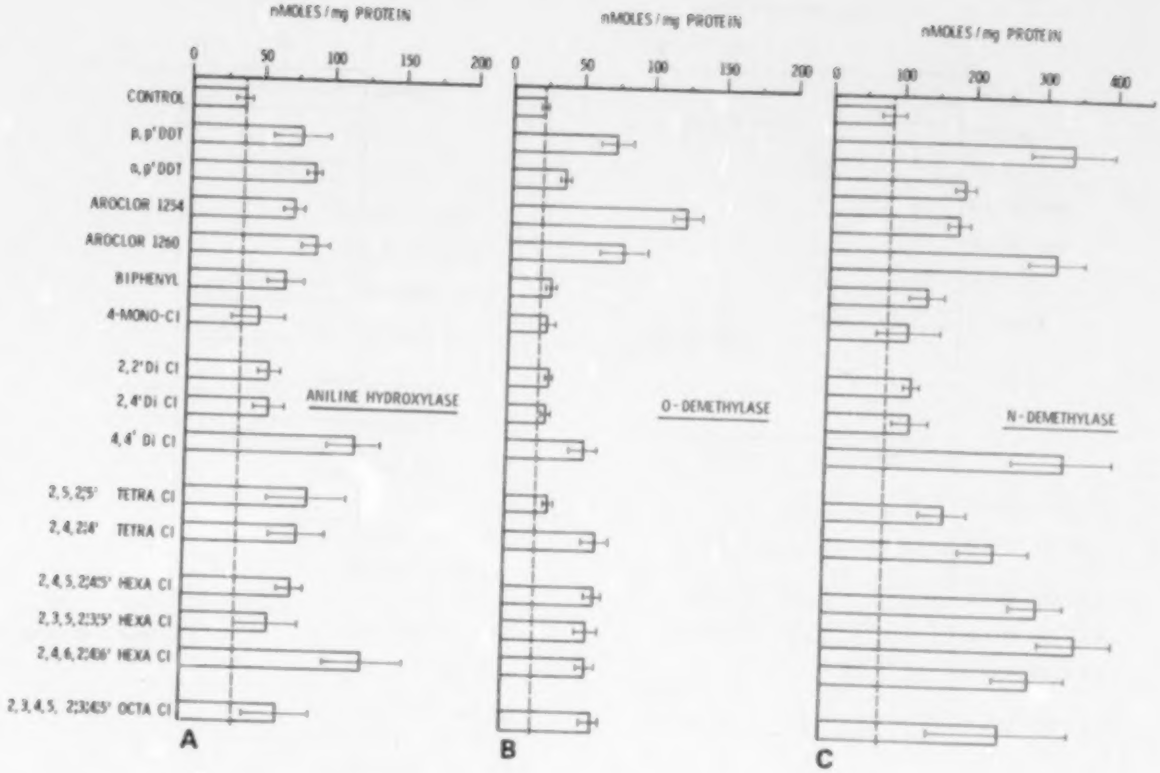

Figure 4. The effect of pretreatment of young male rats with DDT (o,p'- and p,p'-isomers), Aroclor 1254 and 1260, biphenyl, and a series of isomerically pure chlorobiphenyls on hepatic microsomal aniline hydroxylase (A), p-nitroanisole O-demethylase (B), and aminopyrine N-demethylase (C), activities. Activities are expressed as nmoles of product formed/mg microsomal protein/30min incubation. For other details, see figure 2.

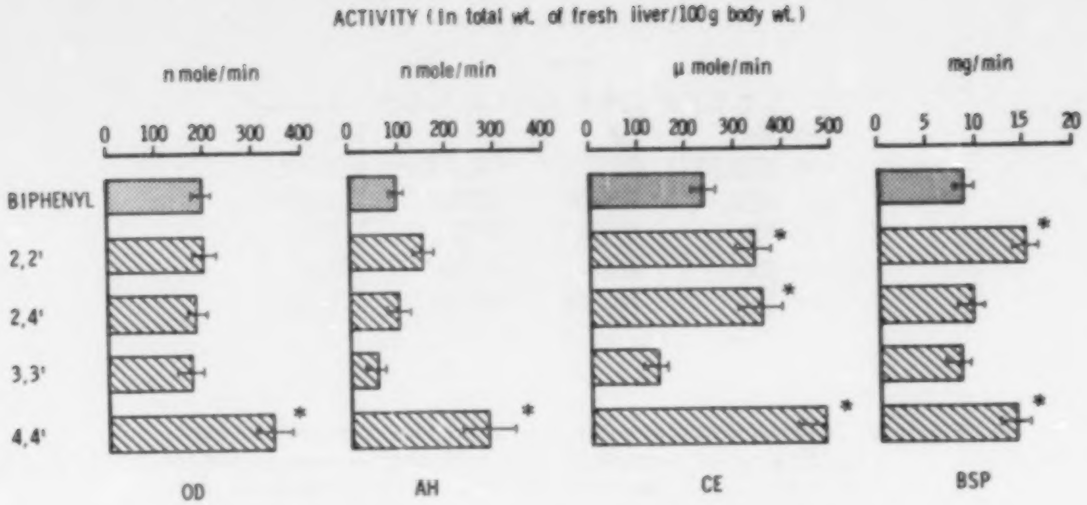

Figure 5. The effect of pretreatment of young male rats with biphenyl and series of isomerically pure dichlorobiphenyls on hepatic p-nitroanisole O-demethylase (OD), aniline hydroxylase (AH), carboxylesterase (CE), and solfobromophthalein-glutathione conjugating enzyme (BSP) activities. Animals were treated by i.p. injection for 3 consecutive days and were killed and assayed 96 hr after the last injection. The values (bars) represent the mean enzymatic activities ± S.D. (lines) of 18 control, biphenyl-treated rats and 6 animals per treated group. The asterisk (*) indicates values statistically different ($p < 0.55$) from biphenyl-treated controls.

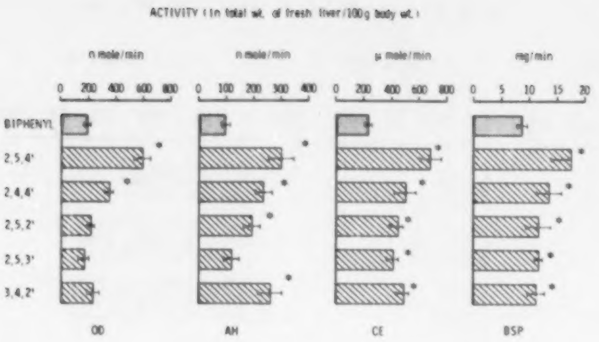

Figure 6. The effects of pretreatment of young male rats with biphenyl and a series of isomerically pure trichlorobiphenyls on hepatic p-nitroanisole O-demethylase (OD), aniline hydroxylase (AH), carboxylesterase (CE), and sulfobromophathalein-gluthathione conjugating enzyme (BSP) activities. For other details, see figure 5.

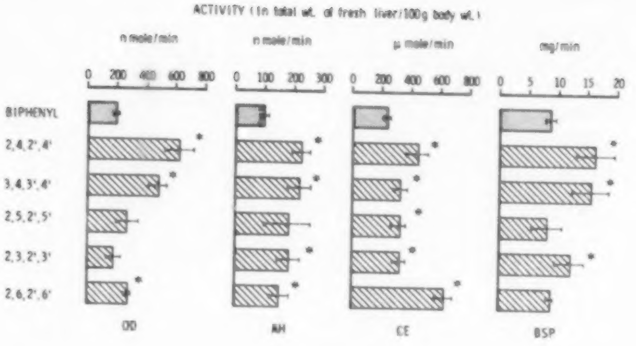

Figure 7. The effects of pretreatment of young male rats with biphenyl and a series of isomerically pure tetrachlorobiphenyls on hepatic p-nitroanisole O-demethylase (OD), aniline hydroxylase (AH), carboxylesterase (CE), and sulfobromophthalein-glutathione conjugating enzyme (BSP) activities. For other details, see figure 5.

biphenyls possess the same toxicologic properties, the position of chlorination being as important as degree of chlorination.

The porphyrogenic nature of chlorinated biphenyls is well known (refs. 2,13-15). Goldstein et al. and Grote et al. demonstrated marked increases in δ-aminolevulinic acid synthetase following treatment with commercial chlorobiphenyls (refs. 15,16). This enzyme, located in hepatic mitochondria, is the rate-limiting catalyst in the synthesis of heme and prophyrins. Recently, Goldstein et al. completed a study of the effects on chick liver, feeding five different, isomerically pure hexachlorobiphenyls at concentrations of 400 ppm or 3 weeks (ref. 17). All agents caused uroporphyrin accumulation, increased hepatic cytochrome $P\text{-}^{450}_{450}$, and microsomal drug-metabolizing enzymes but only 3,4,5,3′,4′,5′-, 2,3,4,2′,3′,4′-, and 2,4,5,2′,4′,5′-hexachlorobiphenyl caused gross accumulation of hepatic porphyrins. These isomers were also the most toxic. It is notable that these three isomers had both the *m*- and *p*-positions occupied by chlorines, while the other two isomers (2,3,6,2′,3′,6′- and 2,4,6,2′,4′,6′-) had either the *m*- or the *p*- positions unoccupied.

Several investigators have demonstrated that the low chlorine-containing congeners in commercial mixtures disappeared more rapidly from tissues than did the highly chlorinated biphenyls (refs. 18-22). In our earliest paper, we suggested that the magnitude of hepatic enzyme induction might be related to the rate of degradation and elimination of the congeners from the body (3). There is ample evidence of hydroxylated derivatives of chlorobiphenyls being eliminated from mammalian systems and, no doubt, we shall hear more about them at this meeting (refs. 23-27). Evidence in the literature suggests two possible mechanisms of biotransformation. The first and most rapid mechanism involves the formation of an arene oxide intermediate and requires the presence of unsubstituted adjacent (vicinal) carbon atoms in the nucleus (ref. 24,27,28). The second and much slower mechanism uses a different hydroxylating system for isolated unsubstituted positions as are found in highly chlorinated biphenyls (ref. 26). The position of chlorination in low chlorine-containing biphenyls could have considerable directing influence over the pathway of biotransformation. Certain low chlorine biphenyls would be less effective (and less toxic) due to rapid biotransformation via arene oxide intermediates. Others, substituted at a position (i.e., 4-position) which would disrupt the vicinal carbon arrangement, would be unable to undergo rapid biotransformation and would persist in vivo. The toxicity of these agents may be closely correlated with lipid solubility, high concentration in the liver, and along duration of action, but it is evident that the position of the substituent chlorines on the biphenyl nucleus may be the key factor governing these other properties.

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DISCUSSION

DR. JOHN V. MOORE (National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina): Have you done any investigatory work to see what the name of the impurity might be?

DR. ECOBICHON: Not that I know of. I don't even belive Eastman Kodak is aware of it.

TOXICOLOGY OF SELECTED SYMMETRICAL HEXACHLOROBIPHENYL ISOMERS: I. BIOLOGICAL RESPONSES IN CHICKS AND MICE

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Abstract

One-day-old cockerels were fed: (I) 3,10,30,100, and 300 ppm of 3,4,5,3',4',5'-hexachlorobiphenyl (HCB); (II) 400 ppm of 2,3,4,2',3',4'-HCB; (III) 400 ppm of 2,4,5,2',4',5'-HCB; (IV) 400 ppm of 2,3,6,2',3',6'-HCB; and (V) 400 ppm of 2,4,6,2',4',6'-HCB. Surviving chicks were sacrificed at 21 days. Male mice were fed 10, 30, 100 and 300 ppm of three of the above isomers (I, III, V), and survivors were sacrificed at 28 days. HCB's levels in adipose tissue and liver were determined. There were variations among the isomers as to dose and pathologic effects. Isomer (I) showed the greatest effect of those studied on mortality, body weight gain, liver, thymus, and spleen; it also attained the highest tissue concentration. It was the only isomer which produced porphyrin accumulation; and, in the chicks, produced hydropericardium, ascites, and edema. The decreasing order of overall toxicity was I >> V > II, III, IV. A general similarity of response was observed in both chicks and mice.

INTRODUCTION

A number of publications have described various toxicologic effects of commercial polychlorinated biphenyls. The presence of toxic impurities, variable chlorine content, or chlorine substitution patterns make interpretation and comparison of the results of these studies quite difficult.

By comparison, little work has been done on assessing the general toxic effects of single polychlorinated biphenyls. The objective of this report is to describe the first results obtained from a comparative, systematic study of the general biological effects of some hexachlorobiphenyl (HCB) isomers in chicks and mice.

The HCB's were selected because of (1) their predominant presence in higher chlorinated formulations (ref. 1); (2) their relative stability in the environment (particularly their persistence in human tissue) (ref. 1); and (3) their strong inductive effect on several biological parameters (ref. 2). The specific isomers studied were selected as biphenyl models representing different physicochemical properties. Chicks were used in this experiment because of their high sensitivity to polychlorinated hydrocarbons; mice were selected to confirm results in a mammalian species.

MATERIALS AND METHODS

The experimental protocol is summarized in table 1. The table illustrates the chemicals and dose levels used, the number of animals per dose level, and the duration of the experiment. The HCB's were mixed in a chick edema bioassay diet or a standard powdered mouse diet. The animals were housed in temperature- and humidity-controlled rooms. The chicks were kept in wire-floored cages, the mice in separate plastic cages. Food and water were provided *ad libitum* for the entire experimental period. The animals were observed daily; body weight was recorded twice a week; food consumption was measured once a week for the chicks and three times a week for the mice. Control groups were observed under identical experimental conditions.

Complete necropsies were performed on dead or moribund animals and on the surviving animals at the end of the experimental period. Blood samples were obtained at this time and analyzed for packed cell volume, total serum protein, and protein fractionation. Liver, spleen, and heart weights were recorded for the chicks; in mice, kidneys, right testicle, and adrenals were also weighed. All organs and tissues were examined under UV light for the presence of red fluorescence as an indication of porphyrin accumulation. After fixation, tissues were prepared for histopathologic examination using standard methods.

All animals were assigned to a given dose according to a table of random numbers. Diet, adipose tissue, and liver were collected for HCB's residue analysis.

RESULTS

The complete results of this study are described in a series of papers either in press or in preparation (refs. 3-6). This presentation is a summary comparison of major biological responses observed in chicks and mice. Table 2, which summarizes the chick effects, shows that 3,4,5,3',4',5'-HCB is the most toxic isomer studied, causing death in all the chicks, even at the lowest dose used. You will note that this dose is less than 1/100 of the diet concentrations of the other HCB's. Only one out of ten

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chicks died in the 2,3,6,2′,3′,6′-HCB group; no animals of the other groups died during the experiment. Reduction in body weight gain was observed in all treatment groups.

At the dose levels studied, significant differences in liver weights and histopathologic changes were observed with all HCB isomers. The most toxic was 3,4,5,3′,4′,5′-HCB, even at the lowest dose. It was the only isomer in which UV fluorescence (porphyrin accumulation) was observed. This finding was quite pronounced, especially in the lining layer of the gizzard, liver, and bones. The slight pathologic effect of the liver observed at 10 ppm is probably due to the very early death of the chicks. The thymus of these chicks was extremely involuted while only slight effect was caused by the other isomers.

Table 3 summarizes the major pathological changes observed: Marked edema of subcutaneous tissue, denoted by gelatinous appearance of subcutaneous fat; marked depletion of lymphocytes in the spleen; diarrhea and soiling of the cloacal area; turbid ascitic fluid; hydropericardium; and loss of visceral fat were findings seen only with the 3,4,5,3′,4′,5′-HCB. Fatty metamorphosis and single-cell or focal necrosis of the liver, were present, albeit to a variable extent in all animals. In addition, characteristic large black spots, 1 to 5 mm in diameter, were observed under the capsule and on cut surfaces of the liver of birds which received 2,4,6,2′,4′,6′-HCB. This change was caused by marked dilatation of sinusoids with a result of accumulation of blood. In some cases, the epicardium of chicks fed 2,3,4,2′,3′,4′-HCB was markedly edematous.

Table 4 summarizes the effects in mice. Again, 3,4,5,3′,4′,5′-HCB was the most toxic, causing one death at 30 ppm and decreasing body weight gain at the 10-ppm level. Although there were no deaths at 10-ppm level in this experiment, which lasted to the 28th day, in one subsequent experiment, mice fed a diet with 10 ppm died an average of 39.4 days (range 36-47). Only one mouse fed the highest dose (300 ppm) of 2,4,5,2′,4′,5′-HCB died before the end of the experimental period; in contrast, all mice fed 2,4,6,2′,4′,6′-HCB at the same level died.

The body weight gain of the mice fed 100 ppm of 2,4,5,2′,4′,5′-HCB or 2,4,6,2′,4′,6′-HCB were not significantly reduced. The dose-related increase in liver weight was caused by all three isomers, with the greatest effect being in the 3,4,5,3′,4′,5′-HCB group. The absence of liver pathology in the mice fed with 300 ppm 3,4,5,3′,4′,5′-HCB is, again, considered to be related to the early time of death. Only 3,4,5,3′,4′,5′-HCB caused porphyrin accumulation and a dramatic atrophy of the lymphatic organs.

The major pathological changes observed in the mice are summarized in table 5. Gelatinous appearance of subcutaneous fat, presence of blood in the gastrointestinal tract, and hemmorrhage in the retrobulbar area of the eye were characteristic of the mice that died due to 3,4,5,3′,4′,5′-HCB toxicity. Accentuation of the hepatic lobules, swelling and hyalinization of the hepatocytes, fatty metamorphosis, and single or focal necrosis were not related to a particular chemical since these changes were observed in all HCB groups to various degrees. Cardiomyopathy and passive congestion of the lung were caused only by 2,4,6,2′,4′,6′-HCB at the 300-ppm dose.

Retention indices of the mixed liquid phase used for gas chromatography may serve as an index of lipophilicity (table 6). Except for 2,4,6,2′,4′,6′-HCB, decreasing indices for this isomeric series correlated exactly with decreasing adipose tissue accumulation in chicks. The highest values were shown by 3,4,5,3′,4′,5′-HCB and 2,3,6,2′,3′,6′-HCB the lowest. A similar trend in tissue accumulation and retention indices is evident in mice (table 7), but not at all dose levels. A greater concentration of 3,4,5,3′,4′,5′-HCB was observed in all cases, particularly in the liver.

CONCLUSIONS

There are definite differences in the toxicity of the HCB isomers tested. Comparing toxicity using such biological parameters as mortality, body weight gain, liver effects, porphyrin accumulation, involution of the lymphatic organs, and fluid accumulation in chicks, the decreasing order of overall toxicity would be 3,4,5,3′,4′,5′-HCB >> 2,4,6,2′,4′,6′-HCB > 2,4,5,2′,4′,5′-, 2,3,4,2′,3′,4′-, 2,3,6,2′,3′,6′,-HCB.

The differences in pathologic effects observed during these experiments were quantitative and, to some degree, qualitative. It is important to note that the nature of the toxicity of 3,4,5,3′,4′,5′-HCB mimics the effects caused by the dibenzofurans.

Although certain toxicologic characteristics differentiate the biological responses in each species tested, the major toxicopathologic effects were common to both chicks and mice.

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Table 1. Protocol for hexachlorobiphenyl isomers toxicity experiments in mice and chicks[a]

Chemical	Dose level (ppm)		No. of animals/ dose level		Experimental period (day)	
	Mice	Chicks	Mice	Chicks	Mice	Chicks
(>99 percent purity)						
3,4,5,3',4',5'-HCB	3 10 30 100 300	3 10 30 100 300	5	10	28	21
2,3,4,2',3',4'-HCB	-	400	-	10	-	21
2,4,5,2',4',5'-HCB	10 30 100 300	400	5	10	28	21
2,3,6,2',3',6'-HCB	-	400	-	10	-	21
2,4,6,2',4',6'-HCB	10 30 100 300	400	5	10	28	21

[a] Five-week old C57BL/6 male mice; 1-day-old white leghorn cockerels.

Table 2. Summary of the biological effects of hexachlorobiphenyl isomers in chicks

Chemical	Dose level (ppm)	Mortality	Body weight gain (% of control)	Liver effect	Porphyrin Accumulation	Thymus effect
3,4,5,3',4',5'-HCB	3	10/10	-	+++	-	+++
	10	10/10	-	++	+++	+++
2,3,4,2',3',4'-HCB	400	0/8	68	++	-	+
2,4,5,2',4',5'-HCB	400	0/10	66	++	-	+
2,3,6,2',3',6'-HCB	400	1/10	80	++	-	+
2,4,6,2',4',6'-HCB	400	0/10	83	+++	-	+

Table 3. Summary of major pathological changes in chicks given different hexachlorobiphenyl isomers for 21 days

Chemical	Dose level (ppm)	Liver	Thymus	Spleen	Fluid Accumulation
3,4,5,3',4',5'-HCB	3	Marked; fatty metamorphosis, focal necrosis.	Marked; involution.	Marked; depletion of lymphocytes.	Subcutaneous edema, ascites, hydropericardium.
2,3,4,2',3',4'-HCB	400	Moderate; hyalinization, single-cell necrosis.	Slight; Involution.	NS[a]	Epicardial edema.
2,4,5,2',4',5'-HCB	400	Moderate; single-cell necrosis.	Slight; involution.	NS[a]	NS[a]
2,3,6,2',3',6'-HCB	400	Moderate; fatty metamorphosis, focal necrosis, giant cell formation.	Slight; involution.	NS[a]	NS[a]
2,4,6,2',4',6'-HCB	400	Marked; fatty metamorphosis, focal necrosis, giant cell formation, marked focal dilatation of sinusoids.	Slight; involution.	NS[a]	NS[a]

[a] NS = not significant.

Table 4. Summary of the biological effects of hexachlorobiphenyl isomers in mice

Chemical	Dose level (ppm)	Mortality	Body weight gain (% of control)	Liver effect	Porphyrin Accumulation	Thymus effect
3,4,5,3',4',5'-HCB	10	0/5	12	++	+	++
	100	3/5	-	+++	+++	+++
	300	5/5	-	-	-	+++
2,4,5,2',4',5'-HCB	100	0/5	91	+	-	-
	300	1/5	58	++	-	±
2,4,6,2',4',6'-HCB	100	0/5	85	+	-	-
	300	5/5	-	++	-	++

Table 5. Summary of major pathological changes in mice given different hexachlorobiphenyl isomers for 28 days

Chemical	Dose level (ppm)	Liver	Thymus	Spleen	Heart
3,4,5,3',4',5'-HCB	30	Marked; fatty metamorphosis, single-cell necrosis.	Marked; involution.	Moderate; depletion of lymphocytes.	NS[a]
2,4,5,2',4',5'-HCB	300	Slight; swelling of hepatocytes.	Slight; involution.	NS[a]	NS[a]
2,4,6,2',4',6'-HCB	300	Marked; fatty metamorphosis, single-cell necrosis.	Marked; involution.	Moderate; depletion of lymphocytes.	Moderate; cardiomyopathy.

[a] NS = not significant.

Table 6. Gas chromatographic retention indices and tissue hexachlorobiphenyl levels in chicks

Chemical	Dose level (ppm)	GC retention index value	Concentration (ppm)	
			Adipose T.	Liver
3,4,5,3',4',5'-HCB	100	2,820	1,210	-
	300		4,912	
2,3,4,2',3',4'-HCB	400	2,678	3,998	44
2,4,5,2',4',5'-HCB	400	2,542	3,921	59
2,3,6,2',3',6'-HCB	400	2,478	2,893	9
2,4,6,2',4',6'-HCB	400	2,346	4,172	24

Table 7. Gas chromatographic retention indices and tissue hexachlorobiphenyl levels in mice

Chemical	Dose level (ppm)	GC retention index value	Concentration (ppm)	
			Adipose T.	Liver
3,4,5,3',4',5'-HCB	10	2820	508	95
	30		1,383	498
	100		2,278	887
	300		6,912	1,344
2,4,5,2',4',5'-HCB	10	2542	296	7
	30		416	30
	100		1,864	83
	300		3,923	637
2,4,6,2',4',6'-HCB	10	2346	99	6
	30		582	15
	100		1,402	122
	300		4,329	1,022

TOXICOLOGY OF SELECTED SYMMETRICAL HEXACHLOROBIPHENYL ISOMERS: CORRELATING BIOLOGICAL EFFECTS WITH CHEMICAL STRUCTURE

James D. McKinney, Ph.D.*

Abstract

The symmetrical hexachlorobiphenyls (HCB's) represent model PCB's with high and constant chlorine content permitting unequivocal study of a given substitution pattern. These isomers show separate and distinct biological responses which can be related to chemical structure via effects of varying chlorine substitution on compound lipophilicity and metabolism. Compound purity must also be assured. Relative molecular polarizability can be correlated with lipophilicity and biological activity and is measurable by chromatographic and spectroscopic techniques. These parameters were highest in HCB's with planar symmetry and 4,4'-substitution. The rates of metabolism of these isomers are considered of lesser importance than the potentially highly toxic nature of some of the intermediary and terminal metabolites.

INTRODUCTION

A previous paper (ref. 1) has shown the importance of the degree of chlorination of PCB's in terms of their distribution and excretion in the rat. Of particular concern is the implication that not more than 20 percent of the hexachlorobiphenyl isomer studied would ever be excreted. Therefore, hexachlorobiphenyls may represent PCB's with both high chlorine content and biological half life. Our studies with symmetrical hexachlorobiphenyl isomers (HCB's) sought to determine the varying biological effects of HCB's as a function of their varying substitution patterns (figure 1). Two of the isomers studied are found in about 3 to 4 percent (area percent by flame ionization gas chromatography) in Aroclors 1254 and 1260. Several major components of these same Aroclors contain various combinations of the substitution patterns studied. Since the effects of chlorine substitution in biphenyls on spectral and chromatographic properties may be approximately predicted (refs. 2,3) using additive parameters obtained from chlorobenzenes, it may be possible to predict the biological behavior of the various combinations through study of the appropriate individual symmetrical isomers.

TOXICOLOGICALLY SIGNIFICANT PROPERTIES

As previously described (ref. 4), our studies with both chickens and mice have shown separate and distinct biological effects at submaximal doses for the different isomers even though all of these isomers might be considered potent (relative to lower chlorinated isomers). In explaining these differences, we feel that the three major considerations are compound purity, lipophilicity, and metabolism. Intestinal adsorption was not considered since earlier work (ref. 5) failed to show appreciable differences for various PCB's.

Compound purity can be very important since both the individual synthetic isomers (ref. 6) and the Aroclor mixtures are known (ref. 7) to be contaminated with small amounts of chlorinated dibenzofurans which, in some species at least, are orders of magnitude more toxic than the PCB's. Since separate and distinct differences in the biological effects of 2,3,7,8-tetrachlorodibenzofuran and the HCB's were found in our work (refs. 8,9), one may be in a position to assess the involvement of dibenzofurans as contaminants and, as will be described later, as potential metabolites.

Relatively little work (ref. 10) has been done associating the effects of varying PCB structure with varying degrees of tissue accumulation. Our work (ref. 8) does appear to show a trend in adipose tissue accumulation of HCB's which correlated with molecular polarizability as measured by chromatographic and spectroscopic techniques. The HCB gas-chromatographic (GC) retention indices (increasing) generally correlated with their overall biological response and were highest in isomers with 3,4-substitution. The amount of HCB in the adipose tissue does not determine activity, but its distribution in this tissue and activity in the liver may both reflect lipophilicity. Since the GC retention index correlation is consistent with a number and variety of GC columns, it is possible that it may have some value in predicting lipophilicity. The lipophilicity in turn relates to biological activity through effective tissue concentration of a given HCB. The differences in adipose tissue accumulation do not appear to be a function of metabolic removal since the least accumulated isomer (2,3,6,2',3',6') was one of the least metabolized (ref. 11).

Considerations of metabolism must take into account the differences in rates of degradation and excretion as well as the nature of intermediary and terminal metabolites. Higher degradation rates (shorter biological half-life) are generally associated with those PCB's having vicinal (two-adjacent carbon atoms) positions unoccupied. These PCB's are also the only ones for which there is clear evidence to support arene oxide intermediates (refs. 1,12). The toxic properties of these PCB's may have more to do with the reactive nature of such arene oxide intermediates, with the main quantitative difference being associated with varying degrees of lipophilicity.

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The more highly chlorinated PCB's such as the HCB's would show similar differences due to lipophilicity, but would be expected to be metabolized more slowly and by other mechanisms as well since vicinal unoccupied positions occur more infrequently. The probable metabolic pathways in chicks (based on metabolities identified in excreta) for the 2,4,5,2′,4′,5′-, 2,4,6,2′,4′,6′- and 3,4,5,3′,4′,5′-HCB's suggest that several reaction types, including isomerization, reductive dechlorination and oxidation with and without the loss of chlorine, are operating. The detailed methodology for identifying these metabolites will be described in a separate report (ref. 11). Microbial metabolism in the gut cannot be ruled out.

The metabolites of 2,4,5,2′,4′,5′-HCB include the meta-hydroxy compound, which appears to undergo further oxidation to the para-quinol, suggesting that a direct insertion hydroxylation reaction may be operating as reported (ref. 12) for further oxidatiion of 4′-chloro-4-biphenylol to 4′-chloro-3,4-biphenylldiol. However, arene oxide intermediate formation may be possible at 1,2-carbons with one unoccupied and the other occupied with either a phenyl or chlorine substituent probably resulting in concomitant chlorine (or phenyl) migration or dechlorination. Dechlorination does occur since a trace of pentachlorobiphenyl and pentachlorotrihydroxybiphenyl and pentachlorophemylbenzoquinone metabolites are found. These highly oxidized products including the *p*-quinone, which accompanies the *p*-quinol, are more chemically and perhaps biochemically reactive. Such products could be more toxic as a result of their increased reactivity unless they are rapidly excreted. The quinone form may not be rapidly excreted since it cannot undergo conjugation.

The metabolites identified in the excreta of chickens fed 2,4,6,2′,4′,6′-HCB contaminated food include 2,4,6,2′,3′,4′-HCB and a trace of 2,4,6,2′,3′-PCB, which are accompanied by two compounds whose low-resolution mass spectral and chromatographic properties suggested hexachlorodibenzofuran and pentachlorodibenzofuran structures. High-resolution mass spectral analysis of the suspected hexachlorodibenzofuran metabolite showed that its molecular formula was $C_{13}H_6Cl_6$ and, therefore, was a methylhexachlorobiphenyl or the mass equivalent chloromethylpentachlorobiphenyl. Methyl ethers and esters are conceivable metabolites of PCB's, but this type of metabolite is unexpected and suggests a direct methylation (or chloromethylation) of an aromatic ring in the biphenyl molecule.

High-resolution mass spectral analysis of the suspected pentachlorodibenzofuran is in progress. However, preliminary low-resolution gas chromatography-mass spectral data obtained from perchlorination of the metabolite mixture indicated that some octachlorodibenzofuran was present. In the absence of the high-resolution data, it is conceivable that the suspected pentachlorodibenzofuran is the source of the octachlorodibenzofuran.

Data obtained from spiking of control chick feces with the 2,4,6,2′,4′,6′-HCB and performing the same extraction and cleanup procedure used for the chick metabolites cast doubt on the authenticity of the pentachlorobiphenyl metabolite and on an additional metabolite corresponding in molecular formula (by high-resolution mass spectral analysis) to $C_{12}H_4Cl_4O_2$ found in liver tissue and later in the excreta of the chicks fed the 2,4,6,2′,4′,6′-HCB.

It is clear from the structures of some of these metabolites that chlorine removal and migration have occurred. Therefore, these products may be derived from enzymatic one electron reduction of this system followed by loss of chloride ion to generate a reactive radical which can react directly with nucleophilic constituents or undergo a second electron reduction to an anion which can react directly with electrophilic constituents. Both electrophilic and nucleophilic radicals of hydrogen (dechlorination), chlorine (isomerization), and oxygen (oxidation) are among the possible recombinants which could account for the metabolites found. The methylhexachlorobiphenyl could also be the result of interaction with an electrophilic methyl donor. Perhaps of equal or greater toxicological significance would be the nature of nonexcretable metabolites derived from covalent bonding of these reactive radicals.

The propensity to lose chloride ion (ref. 13) stems from the favorable stereoelectronic properties associated with a high degree of ortho substitution. The possibility that any oxygenated products including dibenzofurans are the result of direct superoxide anion displacement of chlorine cannot be ruled out. Recent work (ref. 14) has shown that chlorine displacement by superoxide can take place in the mass spectrometer under negative chemical ionization conditions. An analogy to this and possibly an environmental source of dibenzofurans from PCB's would be photonucleophilic displacement of chlorine by water (ref. 15).

In contrast, the 2,3,6,2',3',6'-HCB was metabolized to a trace amount of a monohydroxy metabolite, but no other PCB's or other oxygenated products were found. Therefore, four ortho chlorines alone are not enough; it may be necessary to have high lipophilicity (4,4'-substitution) as well as vicinal unoccupied positions available for appreciable metabolism.

No metabolites were detected in the excreta of chickens fed food contaminated with the 3,4,5,3',4',5'-HCB. However, this isomer has separate and distinct biological effects indicative of a chlorinated dibenzofuran. Since this isomer has a high lipophilicity and accumulation in tissue, its slow metabolism to an elusive, but potentially highly toxic, dibenzofuran may be significant. Enzymatic oxidation at any one of the equivalent unoccupied ortho positions could lead via rearrangement to a "transannular epoxide" intermediate which would undergo a rather facile oxidation to the symmetrical 2,3,4,6,7,8-hexachlorodibenzofuran.

Examination of chicken tissue and mouse excreta and tissue for metabolites is in progress. Preliminary work with tissue from 2,4,5,2', 4',5'- and 2,4,6,2',4',6'-HCB treated chicks has confirmed the presence of tissue metabolites. Metabolites found in tissue are also found in excreta, but not all the metabolites found in excreta are in tissue. At least one metabolite found in the liver and adipose tissue of the 2,4,5,2',4',5'-HCB and in the liver and excreta of the 2,4,6,2',4',6'-HCB treated chicks was of particular interest, since its mass spectral properties (low- and high-resolution measurements) corresponded to a tetrachlorodibenzodioxin (TCDD), a hydroxytetrachlorodibenzofuran, a tetrachlorophenylbenzoquinone or a methoxytetrachlorobiphenyl. The exact structure assignment cannot be made at this time, but failure to methylate this metabolite under two sets of reaction conditions would cast doubt on the hydroxytetrachlorodibenzofuran possibility. As mentioned earlier, this compound may not be a metabolite, but it remains a potential problem as an artifact of the extraction and cleanup procedure for certain PCB's, which could interfere in analysis for TCDD in environmental samples.

SUMMARY

High compound lipophilicity is clearly a contributing factor to HCB toxicity in both chickens and mice. The potentially highly toxic chlorinated dibenzofurans are a problem since they are known to be contaminants of certain Aroclor mixtures and at least one synthetic isomer and are potential environmental transformation products including metabolites of certain PCB's. The exact chlorine substitution patterns found in these compounds may be of considerable importance toxicologically (ref. 16), as has been the case for the structurally similar chlorinated dibenzodioxins. The requisite substitution patterns for dibenzofuran formation found in components of Aroclors most likely would include a combination of those providing high chlorine content, in particular ortho chlorine, high lipophilicity and tissue accumulation, and unavailability of unoccupied vicinal positions. Other PCB's having all ortho positions occupied might also favor ortho oxygenation. Other PCB's may be metabolized to a high degree of ortho-substituents, one of which is hydroxy and another chlorine, which through further metabolism could lead to dibenzofurans, again provided that other requirements are met. Further study is needed with the appropriate purified isomers to clearly establish the formation of dibenzofuran metabolites and determine if microsomal enzymes are responsible.

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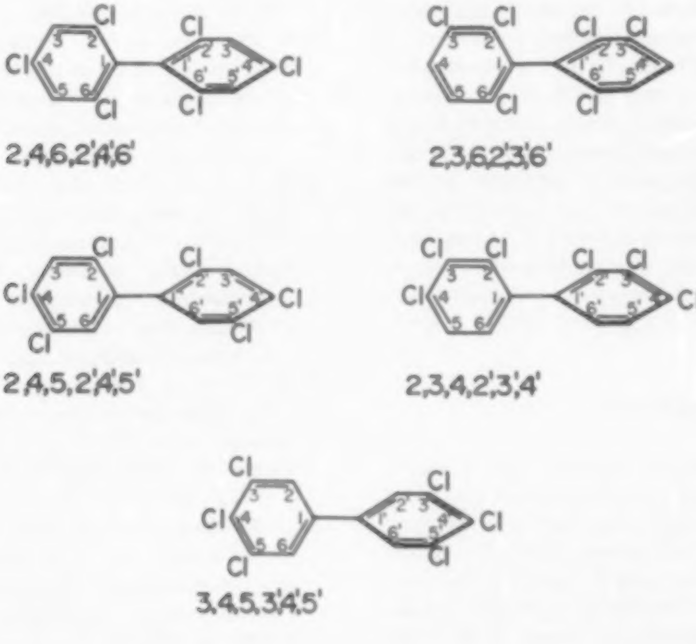
DISCUSSION

DR. STEVEN SAFE (Department of Chemistry, University of Guelph, Ontario, Canada): You mentioned the requirement for 1,2-carbons unsubstituted for arene oxide formation and hydroxylation. We have observed both chlorine migration (NIH shift) and dechlorination. So you do not need unsubstituted positions but you can have a shift.

DR. McKINNEY: I do not believe I said it that way. I believe I said that the only clear evidence that I have seen for oxides has been in cases where adjacent carbon atoms were present.

DR. SAFE: With migration of the chlorine?

DR. McKINNEY: Yes, that is an unusual situation.



2,4,6,2',4',6'

2,3,6,2',3',6'

2,4,5,2',4',5'

2,3,4,2',3',4'

3,4,5,3',4',5'

Figure 1. Hexachlorobiphenyl structures.

TOXICITY OF 2,3,7,8-TETRACHLORODIBENZOFURAN—PRELIMINARY RESULTS

John A. Moore, D.V.M.,[*] B. N. Gupta, B.V.Sc., Ph.D.,[*]
and J. G. Vos, D.V.M., Ph.D.[†]

Abstract

There are at present no reports on the toxicity of pure chlorinated dibenzofurans. The paper below describes ongoing research on 2,3,7,8-tetrachlorodibenzofuran (TCDF), the most toxic isomer of the group. The work is based on the premise that due to the similar structures of chlorinated dibenzofurans and chlorinated dibenzodioxins, the nature of response and the level of exposure needed to induce that response may be similar for the two compounds.

Although no conclusions can be drawn yet, the following observations have been made: (1) Heghorn chicks given 5 μg/kg TCDF showed fluid accumulation, enlarged heart, reduced size of thymus and spleen, then death; (2) Hartley guinea pigs showed weight loss, depression, and diminution of thymus and spleen prior to death; (3) Rats and mice had only mild toxicological reactions to a 6,000 μg/kg dose of TCDF.

Trends at this point indicate that TCDF is highly toxic at low doses to chicks and guinea pigs, as is tetrachlorodibenzodioxin (TCDD). For rats and mice, however, the correlation is not strong. Before accurate assessments can be made of the health significance of chlorinated dibenzofurans, the specific structures of their isomers must be elucidated.

The toxicological significance of exposure to chlorinated dibenzofurans is an issue of great interest. Interest has been generated by the identification of chlorinated dibenzofurans as impurities of two European PCB's (refs. 1,2,3); American PCB's (ref. 4); the presumptive findings of dibenzofurans in the urine of rats fed Aroclor 1254 (ref. 5) as well as the identification of chlorinated dibenzofurans in PCB-contaminated rice oil (ref. 6). There are no reports which describe the toxicologic effects of pure chlorinated dibenzofurans.

Several authors have speculated that due to similar chemical structure, the nature of the response and the level of exposure to induce that response could be similar for chlorinated dibenzofurans and chlorinated dibenzodioxins. To determine the role PCB's per se or a chlorinated dibenzofuran contaminant may play in the toxicity of PCB's, and to simultaneously correlate chlorinated dibenzofuran response to chlorinated dibenzodioxins, initial experiments were undertaken using 2,3,7,8-tetrachlorodibenzofuran. The dibenzofuran with chlorines at the 2,3,7, and 8 positions was felt to be the isomer of initial interest given that it is the isomeric configuration that is the most toxic chlorinated dibenzodioxin.

In all of the studies which I am to report upon today, the 2,3,7,8- tetrachlorodibenzofuran (TCDF) used[††] was of 88 percent purity with the remaining components primarily a pentachlorodibenzofuran. The TCDF was dissolved in acetone, which was then further mixed with corn oil. All doses of TCDF were administered by gavage. We have conducted general toxicity studies with TCDF in chicks, mice, guinea pigs, and rats.

In most instances, the data to be presented represent current status of ongoing research; as such, it does not allow for a clear concise conclusion at this time. However, I do feel the data to be sufficient for one to perceive trend, and I present it with this in mind.

In one experiment (ref. 7), TCDF was administered daily by gavage utilizing white leghorn chicks that were 1 day of age when the study commenced. Birds which received 5 μg/kg per day TCDF died in an average of 11.5 days. General unthriftiness and apparent depression preceded death. One of six chicks receiving 1 μg/kg per day TCDF died on day 19. Body weight and food consumption decreased in this group during the 14th to 21st day of the study. The experiment was designed to terminate on the 21st day. It was our impression that had the study endured an additional week, most, if not all, chicks at the 1 μg/kg dose would have died. The most striking gross pathologic change observed was the accumulation of clear fluid, as evidenced by marked subcutaneous edema, ascites, and hydropericardium, with the severity of the fluid accumulation greatest at the 5 μg/kg dose. The hearts of these birds also appeared enlarged and flabby (rounded). The second striking change was a marked reduction in size of the thymus and spleen. Histologically, size reduction could be accounted for by the marked depletion of lymphocytic cell types in the spleen and cortex of the thymus. Similar pathologic changes were found to occur at the 1 μg/kg dose, albeit to a milder extent. There were no significant effects observed on liver weight; mild liver pathology was found to occur only at the 5 μg/kg dose. Total serum protein levels were reduced with a marked reduction in serum albumin.

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[††] Kindly supplied by Dr. A. Kende, University of Rochester, Rochester, New York.

The toxic response described is similar to that observed in "chick edema disease," a syndrome later shown to be produced by chlorinated dibenzodioxins. This TCDF response is in general agreement with the response that Vos observed in birds that received PCB's that contained low levels of tetrachloro- and pentachlorodibenzofurans.

Toxicity studies with TCDF in Hartley guinea pigs are summarized in table 1. This table shows that the single oral LD_{50} dose lies between 5 and 10 μg/kg. It is also evident that there is an inverse relationship between increased dose and decreased mean time to death. The bottom of the table shows, for comparative purposes, that the LD_{50-30} (that dose which causes 50 percent mortality in a 30-day period) of 2,3,7,8-tetrachlorodibenzodioxin (2 μg/kg) compares quite closely with the TCDF value. TCDF toxicity in guinea pigs is one of a progressive weight loss followed by depression at least 24 to 36 hours preceding death. As was observed in the chicken toxicity studies, there was severe diminution in size of thymus and spleen. Similarly, this finding was found to correlate with a lymphocyte depletion in the periarterialor lymphocytic sheaths and follicles of the spleen; or severe atrophy of the thymus cortex. Although the numbers of animals evaluated were small, the trend of changes present in the bladder, adrenal, kidney, and liver were similar to that previously associated with tetrachlorodibenzodioxin toxicity (ref. 8).

A single experiment in which TCDD was administered to rats at doses up to 1,000 μg/kg failed to show any toxic effects. These rats were terminated 4 weeks after dosing. Histopathologic evaluation of tissues from the rats failed to reveal any changes that could be associated with tetrachlorodibenzofuran toxicity.

Table 2 summarizes the TCDF toxicity findings in male C57B1/6 mice. Per os dose levels up to and including 6,000 μg/kg in a single dose failed to produce any observable effects during the ensuing 28 to 30 days. Histopathologic examinations of these animals failed to reveal any effects on thymus, spleen, or other organs, save for the liver. Here, a mild effect with the suggestions of a mild toxicity was observed in mice that received 6,000 μg/kg per os. A definite toxic response was observed in mice that received a 6,000 μg/kg dose administered subcutaneously. Although mortality was not observed, there was a depression in body weight gain during the second to fifth day following dosing. Organ weights recorded at time of necropsy 30 days after the dose had been administered showed an increase in the liver weight as well as liver to body weight ratio.

Table 1. 2, 3, 7, 8-tetrachlorodibenzofuran toxicity in Hartley guinea pigs

Per os dose (μg/kg)	Mortality	Mean time to death
80	5/5	10.8 days
40	5/5	11.6
20	5/5	12.2
15	6/6	12.0
10	9/11	15.5
5	3/11	18.0
0	0/11	-0-

LD_{50} 2,3,7,8-TCDD = 2.0 μg/kg; MTD = 20.6 days.

Table 2. 2, 3, 7, 8-tetrachlorodibenzofuran toxicity in male C57 BL/6 mice[a]

Dose:	0 μg/kg	6,000 μg/kg
Mortality:	None	None
Body weight:	Normal gain	Sl. depression 2nd-5th day
Liver/body weight:	1.513 + 0.05	1.728 + 0.04
Ratio:	5.76 + 0.05	6.86 + 0.06
Thymus/body weight:	0.045 + 0.002	0.017 + 0.001
Ratio:	0.19 + 0.01	0.06 + 0.007

[a] Administered subcutaneously to 6-week-old mice. Animals sacrificed 30 days post-administration. "No effects" per, os, at 6,000, 4,000, 2,500, 1,500, 1,200, 1,000, 800, 600, and 400 μg/kg. LD_{50} 2,3,7,8-TCDD = 200-250 μg/kg.

A more striking finding was the reduction in thymus weight with a concurrent reduction in thymus to body weight ratio. Histopathologic evaluations of tissues from these animals confirmed that the reduction in thymus weight was due to loss of lymphocytic elements. Evaluation of the liver evidenced a clear, but moderate, toxic response characterized by focal areas of single- cell necrosis and pleomorphism. The mouse studies show that there is at least a 30-fold diminution in TCDF toxicity when compared to TCDD. It further indicates, however, that the pattern of the toxicity likely will mimic that which is observed with tetrachlorodibenzodioxin (ref. 9).

The health significance of dibenzofurans in PCB toxicity can only be speculated upon at this time. It is clearly toxic at very low dose levels in the chick and guinea pig. However, studies to date with the mouse and, to a lesser extent, with the rat suggest that toxicity in these species may not be as closely correlatable to the corresponding dibenzodioxin analog. Furthermore, I believe the specific structure of chlorinated dibenzofuran isomers must be elucidated before their health significance can be projected with any reasonable degree of confidence. This opinion is based on work with chlorinated dibenzodioxins where marked variability in toxicity was seen dependent on the specific chlorinated isomer tested (ref. 10).

A final point addresses the tendency to speculate that the 3,4,5,3′,4′,5′-HCB toxicities as seen in mice and chickens are due to TCDF. Though the pattern of pathologic response is similar in both instances, the response which is observed is not pathognomonic for a specific chemical.

These dibenzofuran studies are continuing and are being extended to the rhesus monkey as soon as sufficient 2,3,7,8-TCDF is synthesized. A systematic evaluation of other chlorodibenzofurans is planned.

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19 November 1975

Session II:

USES, SOURCES, AND IDENTIFICATION

David Garrett*
Session Chairman

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INTRODUCTORY REMARKS

David Garrett

This session will deal with the uses, sources, and identification of PCB's, their disposal, reclaiming, and their treatment. All of us here on the podium have been required many times to share or contribute to many meetings involving discussions on PCB problems, and we are on a day-to-day basis directly involved with these problems.

Since discovery and commercialization of these substances with such marvelous commercial properties for which there appears to be no universally perfect substitute, some 500 million pounds have been pumped into our environment. Over this period after some 45 years, considering the PCB's still in service, the quantities destroyed via incineration, and natural dissipative forces, estimates have been given that some 125 to 135 million pounds are still available for biological activities.

Our eminent speakers will address both the quantities and active service life and the quantities still available for biological activity.

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CHARACTERIZATION OF POLYCHLORINATED BIPHENYLS

J. P. Mieure, Ph.D.,* O. Hicks, R. G. Kaley, Ph.D., and V. W. Saeger, Ph.D.

Abstract

Assessment of the environmental impact of PCB products cannot be carried out without a basic understanding of the physical and chemical properties of these mixtures. PCB structure and nomenclature and the approximate composition of Aroclor products are presented. Several physiochemical properties which affect the performance and environmental impact of current dielectric products are described.

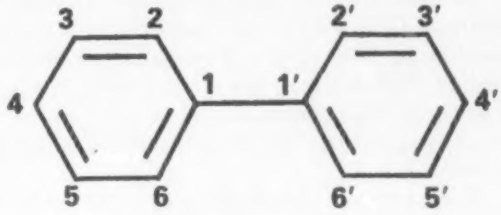
CHEMICAL STRUCTURE

Polychlorinated biphenyls (PCB's) are made by direct chlorination of biphenyl. The biphenyl molecule has 10 possible sites at which chlorine atoms can be substituted. The numbering system for the biphenyl structure is shown in figure 1. By convention, the two rings are numbered identically, except the ring with the fewest chlorine substituents or substituted in the highest numerical positions is designated as primed. Examples of nomenclature for a tri- and a tetrachlorobiphenyl are included in figure 1. An excellent discussion of PCB nomenclature can be found in a recent text by Hutzinger, Safe, and Zitko (ref. 1).

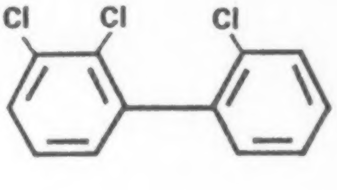
There are 209 possible chlorobiphenyl isomers distributed as indicated in table 1. However, many of these isomers are not likely to occur at significant levels in commercial PCB mixtures. For example, isomers with four or five chlorine atoms on one ring but none on the other ring are not detectable in PCB products.

AROCLOR PRODUCTS

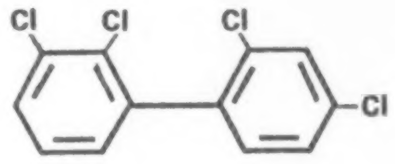
The major producer of PCB's in the United States is Monsanto Company, which markets mixtures for closed electrical system applications under the Aroclor trademark. Aroclor products are identified by a four-digit numbering code in which the first two digits, 12, indicate the parent molecule is biphenyl and the last two digits specify the weight percent of chlorine. The exception is Monsanto's newest product, Aroclor 1016 (41% chlorine), which retained the 1016 designation by which it was known during development. Aroclor products manufactured in the United States are listed in table 2.



BIPHENYL



2,2',3-TRICHLOROBIPHENYL



2,2',3,4'-TETRACHLOROBIPHENYL

Figure 1. The numbering system for the biphenyl structure.

Production of the materials listed as discontinued was terminated in or prior to 1971.

AROCLOR COMPOSITION

The composition of the four Aroclor products currently marketed in the United States is given in table 3. Aroclor 1221 is primarily monochlorobiphenyl while Aroclor 1016 and Aroclor 1242 are primarily trichlorobiphenyls. The major difference between these two products is the lower penta- and hexachlorobiphenyl

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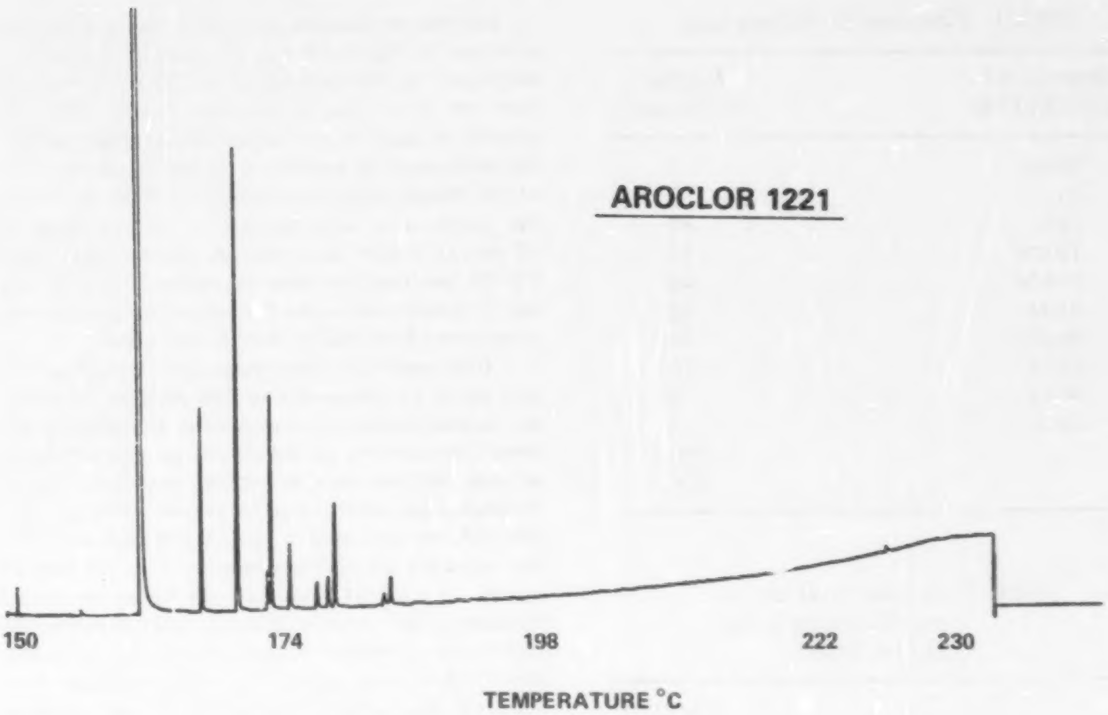

Figure 2. Detailed chromatogram of Aroclor 1221.

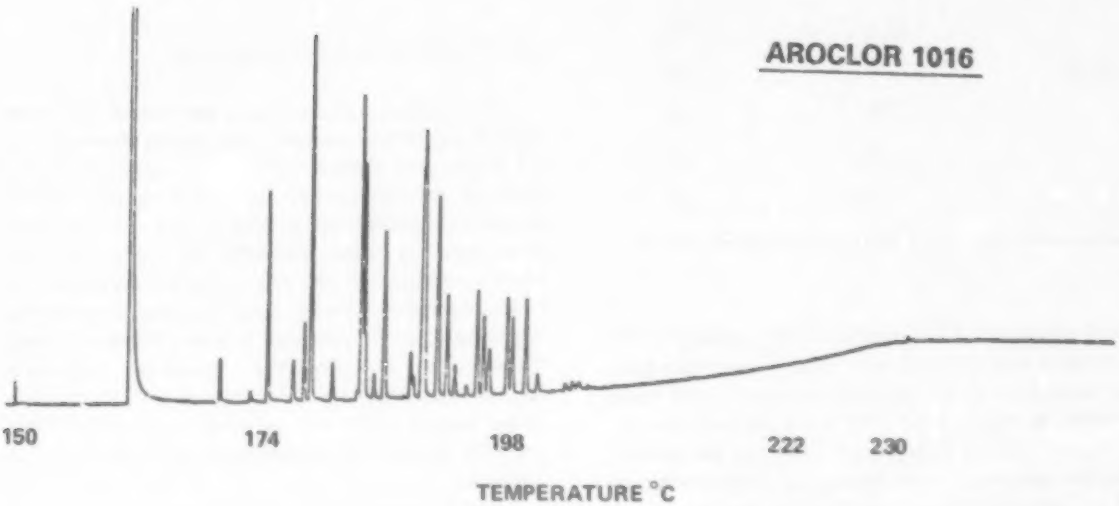

Figure 3. Detailed chromatogram of Aroclor 1016.

Table 1. 209 possible PCB isomers

Degree of substitution	Number of isomers
Mono	3
Di	12
Tri	24
Tetra	42
Penta	46
Hexa	42
Hepta	24
Octa	12
Nona	3
Deca	1
	209

Table 2. Aroclor products manufactured in the United States

Current	Discontinued	Percent chlorine
1221		21
	1232	32
1016		41
1242		42
	1248	48
1254		54
	1260	60
	1262	62
	1268	68

content of Aroclor 1016. Aroclor 1254 is primarily pentachlorobiphenyl. The values in table 3 were calculated from measurements by gas chromatography with flame ionization detection after component identification by gas chromatography/mass spectrometry. All gas chromatographic separations were made with a high-resolution capillary column. Average response factors determined for each degree of chlorine substitution were applied to all components.

Detailed chromatograms of these four products are illustrated in figures 2-5. Specific isomer identifications determined by Webb and McCall (ref. 2) and Sissons and Welti (ref. 3) are listed for the major Aroclor 1242 components in figure 4. The complexity of these mixtures was emphasized by amplifying the first and last portions of the Aroclor 1254 chromatogram in figure 5. Eighty-five components were detected in Aroclor 1254. A .32-mm id x 50-m glass capillary column coated with OV-101 was used for these separations. The flow rate was 2 ml/min helium and the column temperature was programmed from 150° to 230° C at 2° C/min.

High-resolution chromatographic conditions are very useful for characterizing PCB products. However, for routine monitoring of environmental materials, it is more appropriate to use gas chromatography with packed columns and electron capture detection. Typical chromatograms of this type for Aroclor 1016 and Aroclor 1242 are compared in figure 6. The chromatograms are essentially identical for retention times less than 70 relative to p,p′-DDE. However, the higher chlorinated components with relative retention times 70 and greater represent a significantly smaller contribution in the Aroclor 1016 chromatogram. These chromatograms illustrate the feasibility of distinguishing these unaltered products. However, in environmental materials the characteristic features of Aroclor 1242 are frequently obscured by interfering compounds or by component alteration so the distinction is less clear or sometimes entirely lacking. These separations were carried out on a 6 ft x 4-mm id glass column packed with 3 percent SE-30 on 80/100 mesh Chromosorb W-HP at 190° C.

PHYSICOCHEMICAL DIFFERENCES

The physical and chemical properties of PCB isomers and commercial mixtures vary greatly depending on the degree and position of chlorine substitution. The effect of chlorine content on several physicochemical properties is summarized in table 4. Three of these properties, volatility, water solubility, and biodegradability, are very relevant to the environmental occurrence of PCB's. Volatility and water solubility represent potential mechanisms for introduction into and transport within the ecosystem. Biodegradation represents a mechanism for removing PCB's from the environment. Illustrations of the relative significance of these properties for different PCB isomers are evident from the following three examples.

Relative volatilities for the PCB components in Aroclor 1016 are indicated in figure 7 by comparing chromatograms of liquid and vapor composition. The higher

Table 3. Typical percent composition of Aroclor products

Homolog # Cl/biphenyl	Aroclor 1221	Aroclor 1016	Aroclor 1242	Aroclor 1254
0	11	0.1	0.1	0.1
1	51	1	1	0.1
2	32	20	16	0.5
3	4	57	49	1
4	2	21	25	21
5	0.5	1	8	48
6	ND[a]	0.1	1	23
7	ND	ND	0.1	6
8	ND	ND	ND	ND

[a] ND = none detected, 0.01 percent.

Table 4. Physiochemical properties for PCB products

Property	Percent chlorine 21	42	54
Density (g/ml)	1.19	1.39	1.50
Distillation range (°C)	275-320	325-366	365-390
Fire point (°C)	176	None	None
Vaporization rate (mg/cm^2/hr @100 ° C)	1.74	.338	.053
Water solubility (ppm)	>.2	.2	.04
Biodegradability[a]	81	26	15

[a] Percent degradation/48 hr cycle with semicontinuous activated sludge.

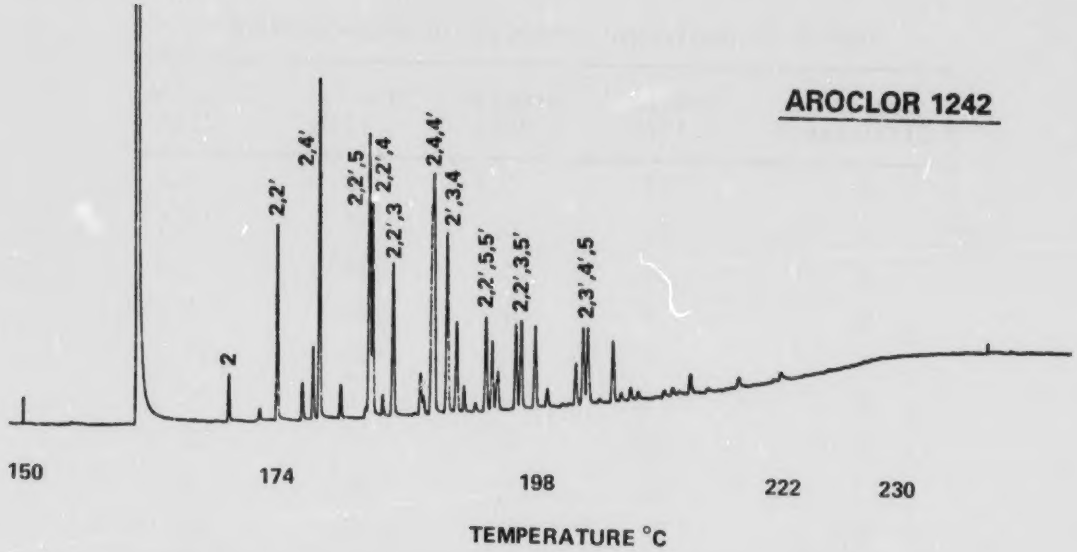

Figure 4. Detailed chromatogram and isomer identification--Aroclor 1242.

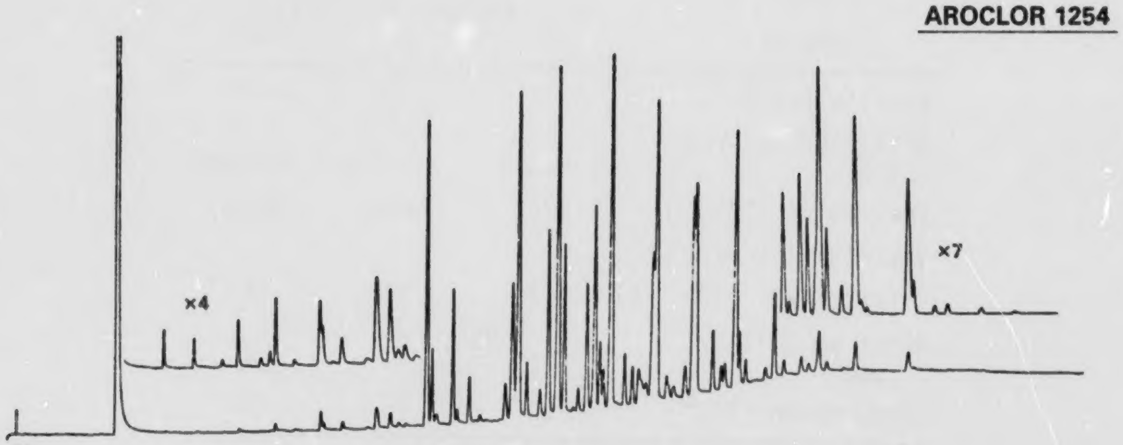

Figure 5. Detailed chromatogram of Aroclor 1254.

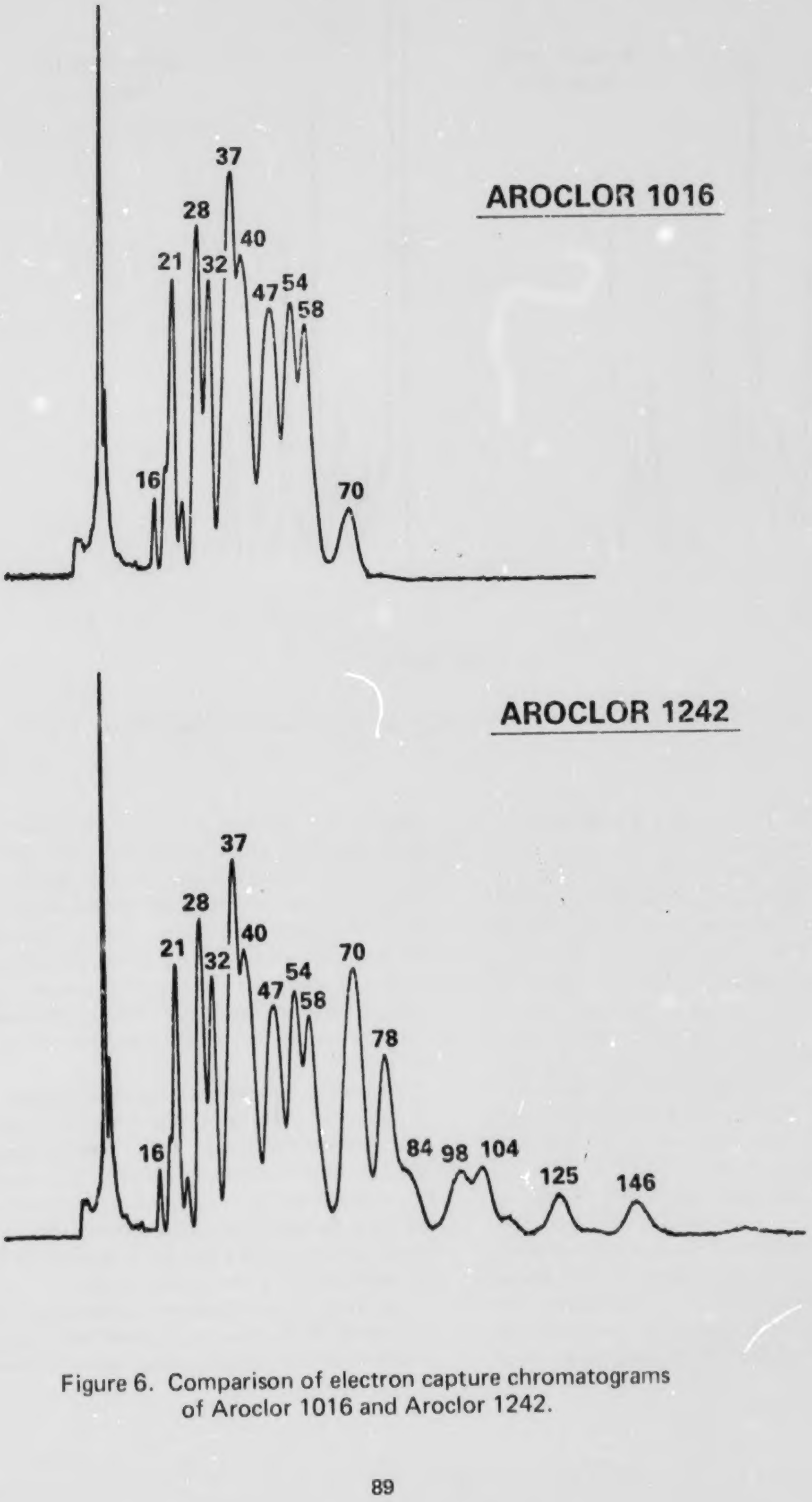

Figure 6. Comparison of electron capture chromatograms of Aroclor 1016 and Aroclor 1242.

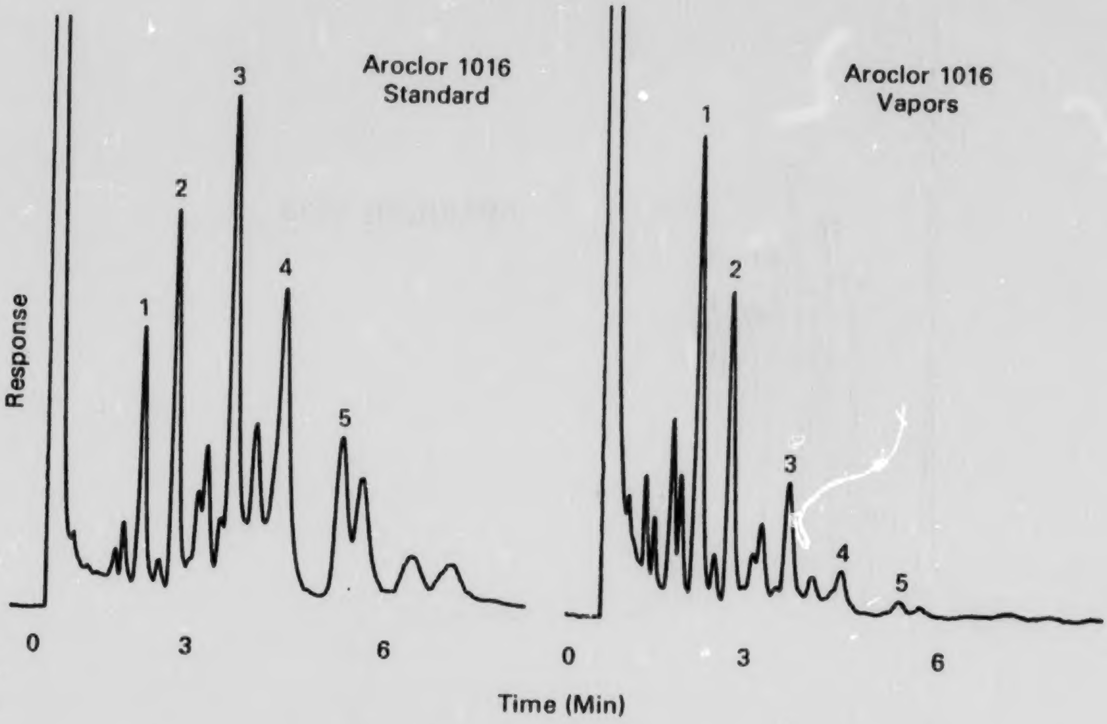

Figure 7. Relative volatilities for PCB components in Aroclor 1016.

numbered components make successively less contribution to the total vapor composition.

Water solubility of Aroclor 1016 components is illustrated in similar fashion in figure 8. Water saturated with Aroclor 1016 was extracted with hexane and the extract compared chromatographically to an Aroclor 1016 standard. The numbers above significant peaks express the percentage contribution of that peak area to the total area. Earlier eluting, lower chlorinated components are more soluble and contribute more to the water saturated composition. A related study has shown that PCB's leaching from Aroclor 1016 impregnated soils after the equivalent of 40 years annual rainfall consist of lower chlorinated biphenyls (ref. 4) (figure 9).

Microbial degradation also affects PCB isomers to different degrees. For example, figure 10 shows chromatograms depicting Aroclor 1016 composition after exposure to a naturally occurring mixed microbial population for 1/2 hour (above) and 14 days (below). The number above each GC peak indicates the percent biodegradation for that component. These percentages range from 17 to >98 and in general are higher for lower chlorobiphenyls. The exposure was carried out with activated sludge from a domestic sewage treatment facility. The sludge was previously acclimated to a mixture of industrial organic compounds before addition of 3 ppm Aroclor 1016. PCB composition was determined by extracting a representative aliquot of mixed liquor with hexane followed by analysis by gas chromatography using a glass capillary column and electron capture detection.

It is apparent from these examples that introduction of PCB's into the ecosystem by volatility or water solubility would enhance the ratio of lower to higher chlorinated biphenyls in the environment. PCB removal from the environment by either of these mechanisms or by microbial degradation would reduce this ratio. These and related factors combine to make assessment of PCB environmental impact a very complex problem.

In view of the recognized complexity in studying commercial PCB mixtures, it would seem expedient to substitute for the mixture a single isomer product having

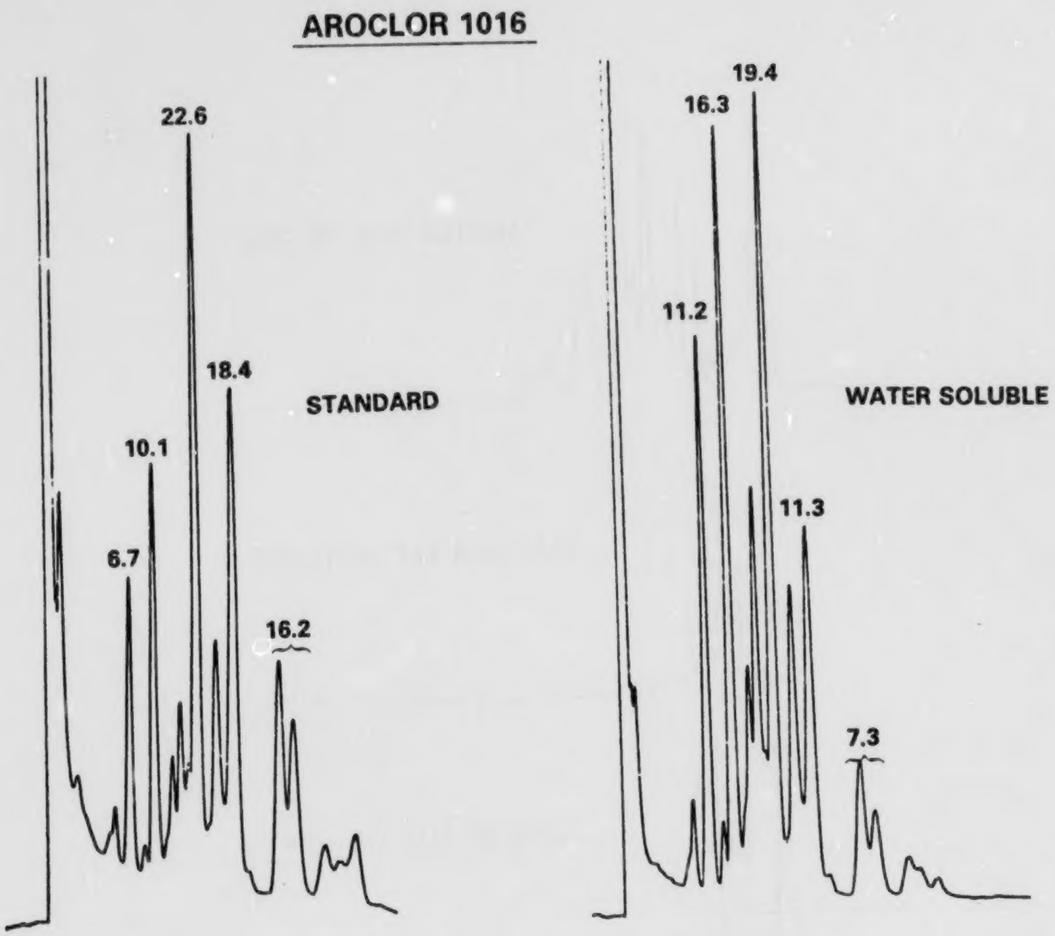

Figure 8. Water solubility of Aroclor 1016 components.

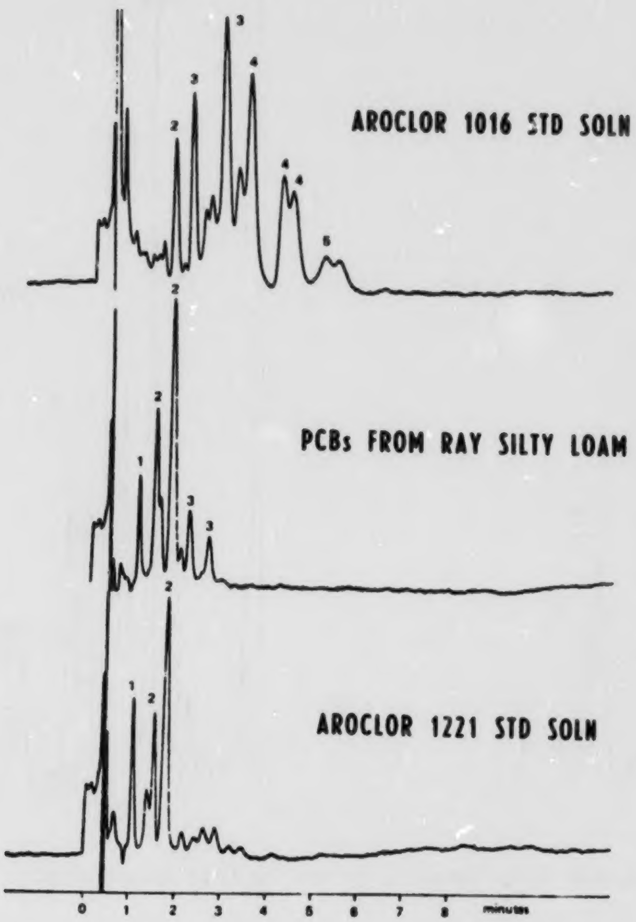

Figure 9. Composition of PCB's leaching from Aroclor 1016 impregnated soil.

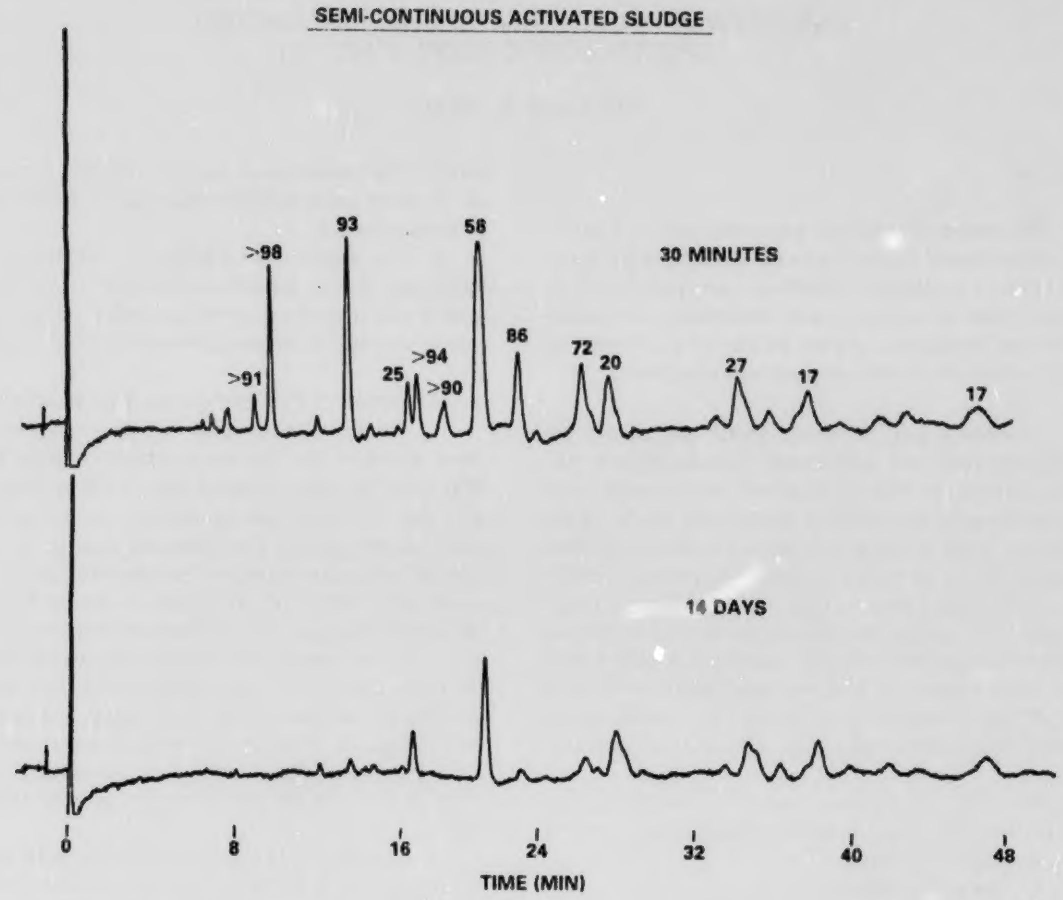

Figure 10. Aroclor 1016 biodegradation.

the desired electrical and physical properties. The environmental properties of this isomer would be much simpler to characterize and control. However, no such PCB isomer exists. Most PCB isomers are solid crystalline materials when isolated at 25° C. Commercial products are liquids only because of the mutual melting point depression exerted by the PCB components of the mixture.

CONCLUSIONS

Commercial PCB products are mixtures of many different isomers with varying chlorine content. The properties of these isomers, and hence the products, are vastly different and are complex to study. The environmental impact for each product must be accurately assessed and compared to the functional benefit.

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OVERVIEW OF ANALYTICAL IDENTIFICATION AND SPECTROSCOPIC PROPERTIES

Stephen Safe, Ph.D.*

Abstract

The analytical problems associated with PCB analysis involve many factors including sampling techniques, extraction and cleanup procedures, quantitation of PCB, confirmation of structure, and identification of metabolites and impurities. Recent studies in these areas of PCB analysis will be discussed and examples given.

Polychlorinated biphenyls (PCB) are among the most persistent and widespread environmental pollutants. Commercial PCB formulations can consist of up to 100 different chlorobiphenyl isomers and this is a complicating factor in the conventional gas chromatographic analysis of environmental samples. The chemical stability of PCB does, however, facilitate their analysis in that a variety of cleanup procedures can be used to remove coextractive material without modification of the PCB. The determination of PCB has been described in detail (ref. 1) and therefore most of the data and results reported herein will be associated with more recent publications.

The analytical problems associated with PCB analysis can be summarized under six major headings, which will be discussed in detail:

1. Sampling methods,
2. Extraction of PCB from environmental samples,
3. Cleanup procedures,
4. Quantitation of PCB levels,
5. Confirmation of structure, and
6. Identification of toxic impurities and metabolites.

1. *Sampling Methods*

PCB analysis is primarily concerned with determination of the pollutant concentrations in biological, sediment (or solid), water, and air samples. Specific sampling methods are required.

a. Biological samples and sediments. The sampling must insure that sufficient material is collected and extracted so that the analytical data obtained are representative of the sample.

b. Water samples. The sampling of water is complicated by the need to distinguish between dissolved PCB, which are usually low, and PCB adsorbed on particulate matter. The separation of the two fractions can usually be obtained using suitable filtration or centrifugation techniques (ref. 1).

c. Air samples. Air samples are usually obtained by passage of a known volume of air through an impinger or a similar device in which a suitable solvent preferentially extracts or removes airborne PCB (refs. 2,3).

2. *Extraction of PCB from Environmental Samples*

a. Biological samples. A number of methods have been described for the semiquantitative extraction of PCB from biological samples (e.g., > 90 percent recovery) (ref. 1). The sample can be macerated then freeze-dried, or dehydrated and extracted directly or by the soxhlet technique using an appropriate solvent. The choice of solvents can vary from nonpolar hexane or pentane to the more polar chloroform, acetone, or methanol. The more polar water-soluble solvents are useful in the extraction of wet tissue; however, the polar aqueous extracts are usually diluted with water and extracted with hexane to preferentially remove the nonpolar chlorinated aromatics. This partitioning technique is also utilized as a cleanup procedure to remove polar coextractives.

b. Sediments. Sediments and other solid samples are usually extracted with more polar solvents and ultimately the PCB are partitioned as described above (ref. 1).

c. Water. The simplest approach with water is by direct extraction with hexane. The removal of PCB from water has also been effected using polyurethane foam plugs and resins (refs. 4-6). Using this technique, the water is passed through a column containing the solid support and the PCB are adsorbed on these supports. Subsequent passage of a polar organic solvent through the columns removes the adsorbed organics, which can be further cleaned up using the partitioning technique.

d. Air. PCB are readily extracted from air by passage of the air through sintered glass funnels or suitably contracted glass impingers filled with solvents (e.g., amyl acetate, sec-butanol or ethylene glycol) (refs. 2,3). The PCB can then be isolated by the standard partitioning technique.

3. *Cleanup Procedures*

The major aim of a cleanup procedure is to selectively remove coextractive natural material and xenobiotics from an extract containing PCB. Solvent partitioning has already been discussed and is an excellent method for removing water-soluble and highly polar coextractive organics. The two additional important cleanup techniques are methods based on column or thin-layer chromatography and chemical treatment of the extract to modify either the PCB or the coextractives.

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a. Chromatographic methods. Most chromatographic procedures include column chromatography on activated florisil and/or alumina using hexane to elute the PCB (refs. 7,8). Thin-layer chromatography with silicic acid and alumina can also be used. An example of a typical chromatographic cleanup procedure is outlined in figure 1.

b. Chemical methods. Since PCB are thermally and chemically stable, it is possible to saponify extracts by both acidic and basic treatment without altering the PCB. This procedure is particularly useful in the conversion of lipid esters, which have chromatographic properties similar to PCB, into more polar products. One of the major problems in PCB cleanup is to remove DDE, which has similar chromatographic properties to PCB. A recent paper describes an oxidative technique which quantitatively converts DDT, DDE, and their analogs into more polar products (ref. 9). Dehydrochlorination with mild base converts DDT and DDT-like compounds into the corresponding bisdiarylethylenes (e.g., DDE); treatment of the olefin with chromic acid gives the corresponding benzophenones, which are readily separated from PCB. The PCB are not affected by this chemical modification procedure. Raney nickel reduction and sulfuric acid treatment of crude PCB extracts are also useful in the cleanup of river water extracts (ref. 10).

The analysis of PCB is always complicated by the large number of isomeric chlorobiphenyls that are present and this results in a complicated gas chromatogram even after cleanup (figure 2). In addition, polychlorinated terphenyls (PCT) and polychlorinated naphthalenes (PCN) are also industrial environmental pollutants which are composed of several isomeric components that can co-occur with PCB. A perchlorination procedure was reported in which the isomeric mixtures can be converted into their respective perchloro derivatives (refs. 11,12), as shown in figure 3. These compounds can then be readily analyzed and quantitated by conventional gas chromatography (figure 2). The comparative gas chromatographic retention times of several perchlorinated pollutants are: decachlorobiphenyl, 1.0; octachlorodibenzofuran, 2.55; octachlorodibenzo-*p*-dioxin, 2.60; dodecachloro-DDE, several peaks > 1.9; tetradecachloro-*o*-terphenyl, 11.9; tetradecachloro-*m*-terphenyl, 19.2; tetradecachloro-*p*-terphenyl, 22.2.

It should be noted that this procedure is markedly dependent on the purity and source of the commercial antimony pentachloride reagent and the composition or degree of chlorination of the PCB. A drawback of this

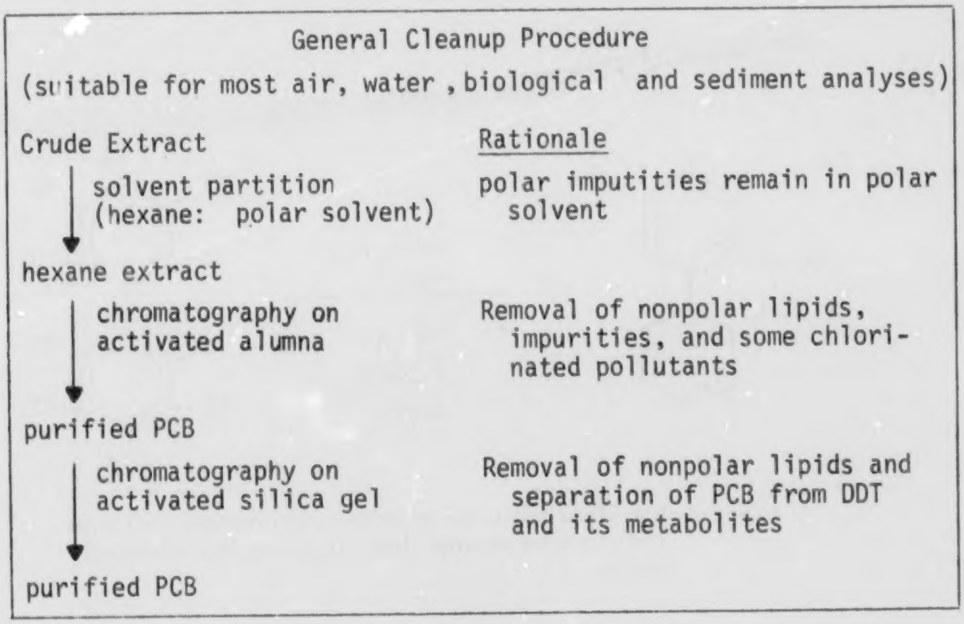

General Cleanup Procedure
(suitable for most air, water, biological and sediment analyses)
Crude Extract
Rationale
solvent partition (hexane: polar solvent)
polar imputities remain in polar solvent
hexane extract
chromatography on activated alumna
Removal of nonpolar lipids, impurities, and some chlorinated pollutants
purified PCB
chromatography on activated silica gel
Removal of nonpolar lipids and separation of PCB from DDT and its metabolites
purified PCB

Figure 1. General cleanup procedure.

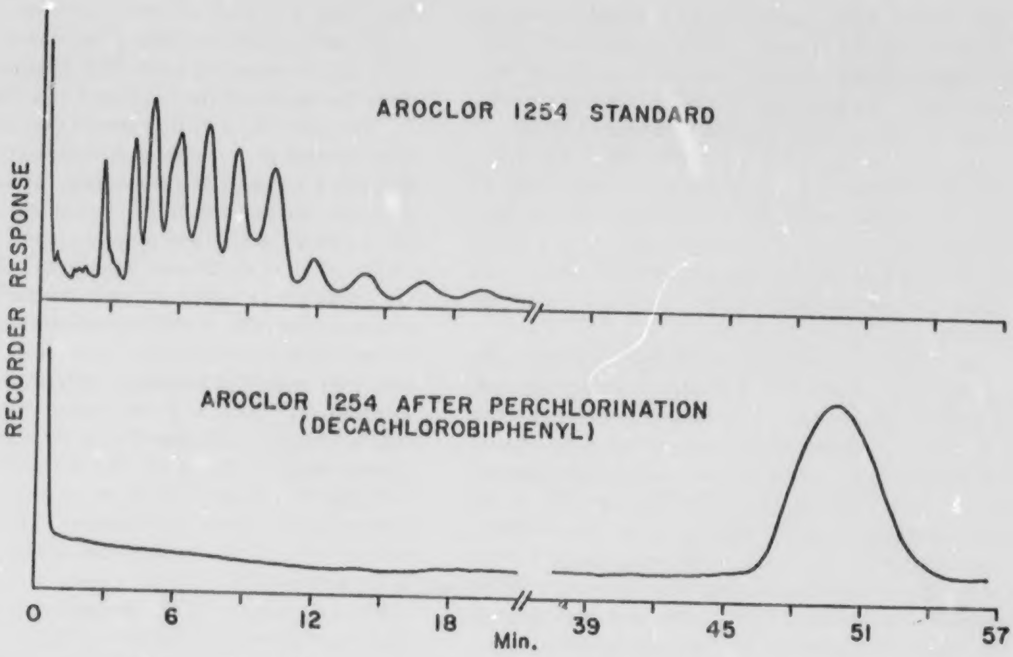


Figure 2. Gas chromatogram after cleanup.

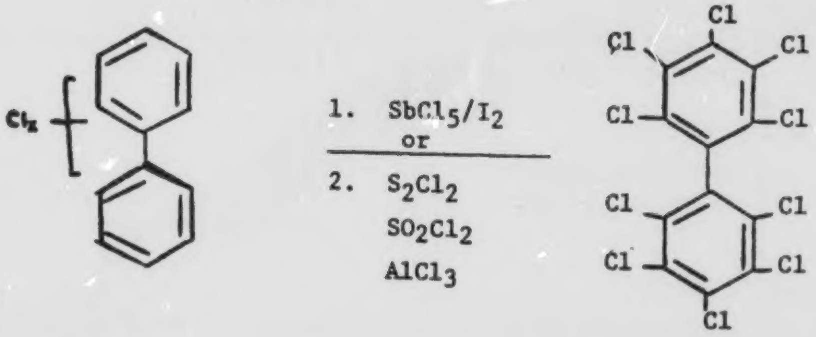


Figure 3. Perchlorination procedure in which isomeric mixtures can be converted into their respective perchloroderivatives.

technique is the reported formation of varying quantities of a sideproduct which has been identified as bromo nonachlorobiphenyl (refs. 13,14). This introduces a second peak which is observed in the gas chromatogram and this side reaction can also reduce the overall yield of the decachlorobiphenyl product.

4. *Quantitation of PCB*

Gas chromatography is the method of choice for PCB analysis and electron-capture detection is by far the most sensitive procedure for detecting these chlorinated aromatic compounds (ref. 1). A comparison of the relative molar responses of electron-capture and flame-ionization detector to some isomeric chlorobiphenyls is given in table 1. It is evident that the electron capture detector is the more sensitive and that the relative molar response is both a function of the degree of chlorination and the position of the substituent (refs. 15,16). Since the precise composition of PCB mixtures is not known, the quantitation of the complex chromatograms can only be an estimated value. A more consistently accurate method would involve perchlorination since the relative molar response and chromatographic properties of decachlorobiphenyl are known. Quantitation of the PCB mixtures is usually obtained by measuring the total peak heights or areas of key diagnostic peaks that are present in the environmental PCB extract and comparing this result with the heights or areas obtained in the gas chromatogram of the appropriate commercial PCB mixture (e.g., Aroclor 1254, Clophen A-60). An example is shown in figure 4, in which the chromatograms of Clophen A-60 and river water extracts are compared and the quantitation is carried out using three diagnostic peaks present in the environmental and commercial PCB sample (ref. 10).

5. *Confirmation of Structure*

PCB in the environment was originally detected by gas chromatography and later confirmed by mass spectrometry and this technique is the most sensitive and

Table 1. Relative molar responses of electron-capture and flame-ionization detectors to some chlorobiphenyls

Chlorobiphenyl	Relative molar response Electron capture	 Flame ionization
2-	1.00	1.00
3-	0.20	0.92
4-	1.10	0.87
2,2'-di	5.16	0.99
2,4'-di	17.7	0.86
2,6-di	32.0	0.91
3,3'-di	6.10	0.94
3,4-di	15.2	0.86
4,4'-di	5.97	0.81
2,4,4'-tri	135	0.78
2,2',4,4'-tetra	106	0.87
2,2',6,6'-tetra	20.6	0.90
3,3',4,4'-tetra	396	0.87
3,3',5,5'-tetra	320	0.85
2,3,4,5-tetra	367	0.87
2,3,5,6-tetra	259	0.71
2,2',4,4',6,6'-hexa	347	
3,3',4,4',5,5'-hexa	726	
2,2',3,3',4,4',6,6'-octa	1180	
2,2',3,3',5,5',6,6'-octa	1150	
deca	1410	

Quantitation of PCB

1. The method of choice is gas chromatography (GC) using an electron capture (EC) detector. The major problem is selecting suitable standard peaks to use as quantitative standards for comparison with the commercial PCB standard samples.

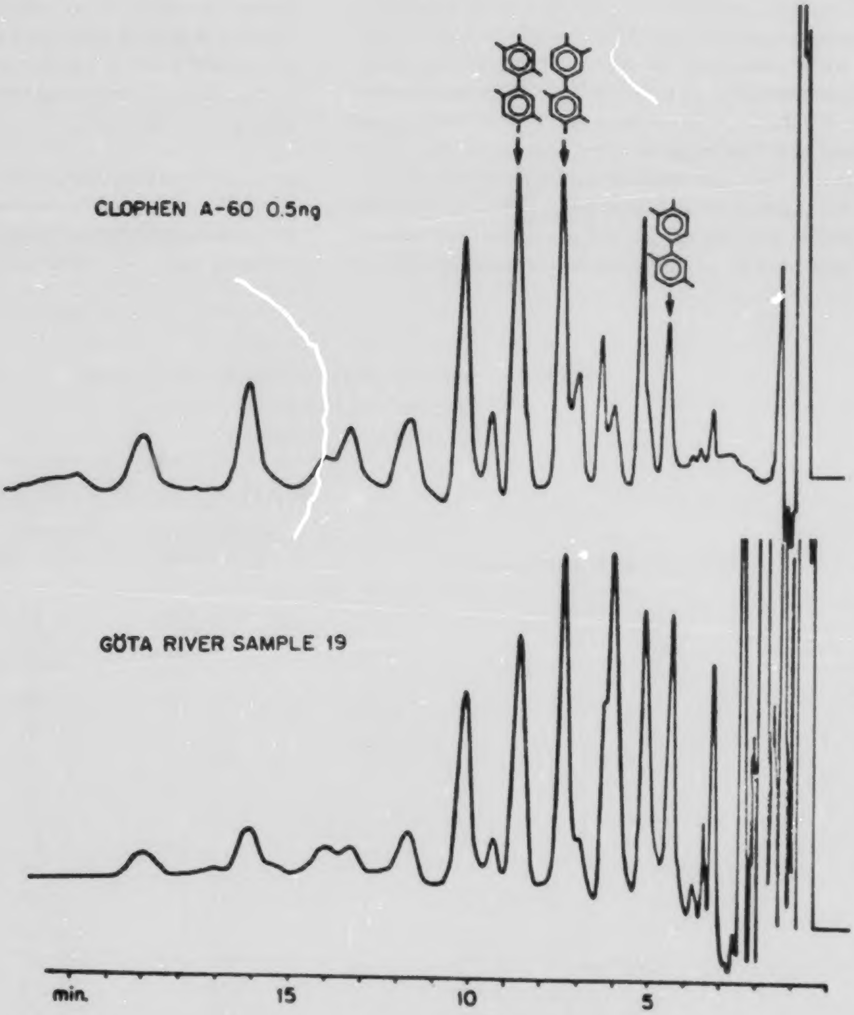


Figure 4. Gas chromatographic analysis with electron capture detector. The injected volume of river water extract corresponds to 1.6 liters water.

widely used spectroscopic method for the confirmation of PCB. The mass spectrometry of isomeric chlorobiphenyls and the applications of this technique in environmental analysis has been reviewed (ref. 17) and only recent applications of this analytical technique will be mentioned.

a. Gas chromatography — mass spectrometry (GC-MS). Gas chromatographic analysis of PCB can be carried out by combination GC-MS so that the mass spectrum of the individual peaks which elute from the gas chromatograph can be recorded. This technique in combination with a computer has been extensively used in environmental analysis for both the detection and confirmation of environmental PCB, related metabolites, and toxic impurities (refs. 1,17). A GC-MS of an environmental sample may generate hundreds of spectra; however, using the computer the analyst can recall individual spectra as well as conduct limited mass searches on key diagnostic ions.

b. Mass fragmentography GC-MS (ref. 18). The mass spectrum of a sample is obtained by scanning the entire mass range in order to record all the ions that are generated. If a mass range of 400 is scanned in a period of 4 seconds, then the mass spectrum is held for only 1/100th of a second at each mass unit; for the remaining 3.99 seconds the ion current at that mass value is lost. Mass fragmentography is a technique which simultaneously monitors the ion current at one or more mass values for the complete duration of the sample spectrum (i.e., for 4 seconds rather than 0.01 second). The mass values which are monitored are the most intense and diagnostic ions generated by a compound. For PCB this is usually the isotopic molecular ion species or the isotopic M-Cl^2 ions. The sensitivity of a GC-MS instrument operating in the mass fragmentography mode can surpass that of an electron capture detector. An example of this technique is shown in figure 5, in which the mass fragmentograms of standard Clophen A-60 and river water

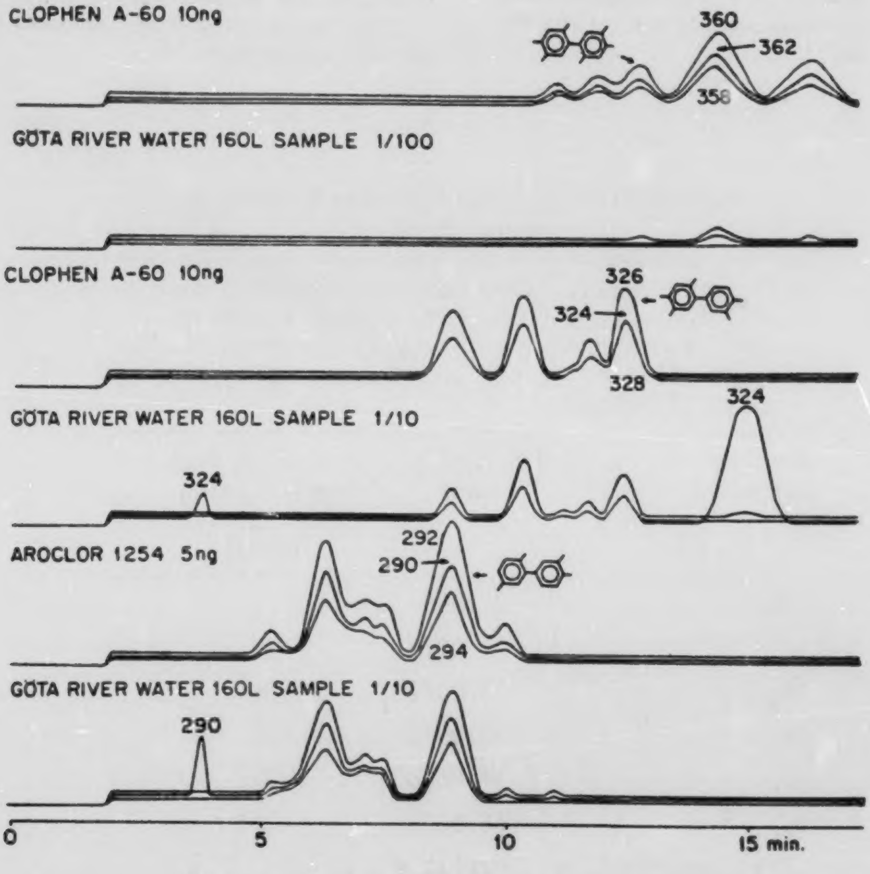


Figure 5. Mass fragmentograms of PCB Standards and river water extracts.

extracts are compared (ref. 19). The diagnostic ions which were used are the isotopic hexachlorobiphenyl (m/e 362, 360, and 358), pentachlorobiphenyl (m/e 328, 326, and 324), and tetrachlorobiphenyl (m/e 294, 292, and 290) molecular ions.

c. High-resolution photoplate mass spectrometry. The relatively large mass deficiencies of both chlorine isotopes cause the exact masses of ions which contain chlorine to be usually less than ions of the same nominal mass that contain only isotopes of C, H, N, and O (see table 2). Thus, a typical lipid coextractive would appear at mass positive integer values whereas the molecular ion for a tetrachlorobiphenyl isomer (accurate mass 289.9224) appears at 776 millimass units below the integer value. Mass negative PCB ions are therefore readily distinguished from mass positive ions which occur at the same nominal mass by high-resolution techniques. A photographic plate can be used as an integrating ion detector and the subsequent identification of the PCB on the plates is readily carried out. This technique has been used in the detection of PCB in biological samples as well as confirming the presence of hydroxylated PCB metabolites (refs. 20,21).

6. *Identification of Toxic Impurities and PCB Metabolites*

Analysis of commercial PCB preparations showed that higher chlorinated naphthalene isomers were present as trace impurities and their structures were confirmed by mass spectrometry (ref. 22). More recently the highly toxic chlorinated dibenzofurans have also been identified in commercial preparations; the flow chart in figure 6 outlines chromatographic procedures for the isolation of these components (ref. 23).

The metabolism of PCB in diverse animal and microbial systems has been investigated and the results indicate the conversion of these substrates into a range of hydroxylated metabolites (ref. 24). Figure 7 summarizes a composite scheme for the separation and identification of PCB metabolites and it should be noted that all the steps are used in the cleanup procedure only when it is necessary. Recent data from seal extracts has confirmed the presence of hydroxylated PCB in their fatty tissue (ref. 25). These metabolites are therefore a new class of chlorinated aromatic pollutants derived from PCB.

The financial support of Environment Canada is gratefully acknowledged.

Table 2. Confirmation of PCB by high-resolution phosphate MS

Basic principles: Xenobiotics containing mass deficient atoms (e.g., Cl) appear at lower mass values than most "natural coextractives"; these differences can be measured using the above technique.

Atom or compound	Accurate mass	Deviation from integer value (in millimass units)
^{12}C	12.000	-
^{1}H	1.0078	+78
^{14}N	14.0031	+31
^{16}O	15.9949	-51
^{35}Cl	34.9688	-312
^{37}Cl	36.9659	-341
Lipid coextractive	variable	

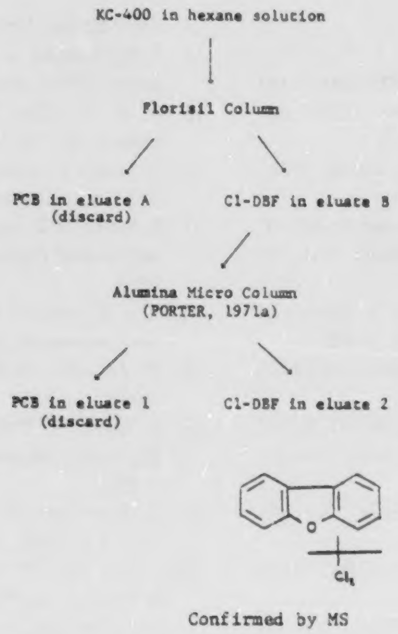
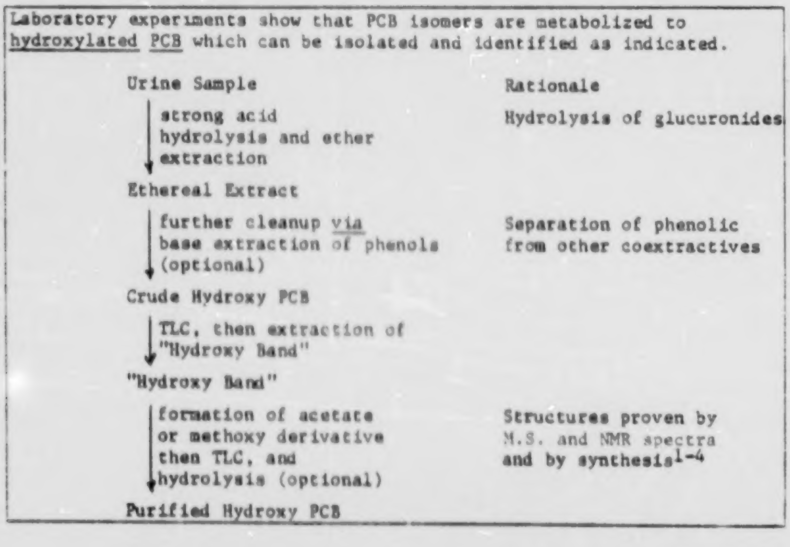


Figure 6. Procedure for separation of chlorinated dibenzofurans from chlorinated biphenyls in KC-400.



Laboratory experiments show that PCB isomers are metabolized to hydroxylated PCB which can be isolated and identified as indicated.

Urine Sample	Rationale
strong acid hydrolysis and ether extraction	Hydrolysis of glucuronides
Ethereal Extract	
further cleanup *via* base extraction of phenols (optional)	Separation of phenolic from other coextractives
Crude Hydroxy PCB	
TLC, then extraction of "Hydroxy Band"	
"Hydroxy Band"	
formation of acetate or methoxy derivative then TLC, and hydrolysis (optional)	Structures proven by M.S. and NMR spectra and by synthesis[1-4]
Purified Hydroxy PCB	

Figure 7. A composite scheme for the separation and identification of PCB metabolites.

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PRODUCTION AND USAGE OF PCB's IN THE UNITED STATES

Robert L. Durfee, Ph.D.*

Abstract

About 99 percent of the PCB's used in U.S. industry are produced by Monsanto in a single facility at Sauget, Illinois. These materials, termed Aroclors, are used in the production of capacitors (70 percent) and transformers (30 percent). Imported PCB's, comprising slightly over 1 percent of U.S. usage, are used in investment casting operations (80 to 90 percent; decachlorobiphenyl) and semiclosed heat transfer applications (10 to 20 percent). Capacitor and transformer production processes are similar in that process steps involving PCB's include filtration, flood or individual filling of the products, and removal of excess PCB's prior to sealing and individual testing of the products. Most losses occur from pump leaks to cooling water, spills, personal hygiene practice, and the like. Many of the discharges containing PCB's are to municipal sewers.

Introduction

Polychlorinated biphenyls (PCB's) represent a class of compounds produced commercially by the chlorination of biphenyl. Most PCB's currently used in the United States are mixtures of chlorobiphenyls containing up to seven chlorine atoms per molecule, although some decachlorobiphenyl (10 chlorines per molecule) is imported. The production and the types of uses for PCB's increased steadily from initiation of production in 1929 until industry-imposed restrictions in 1971 curtailed production and essentially eliminated use of domestic production in all but "closed" electrical equipment (specifically, capacitors and transformers).

PCB's have been increasingly identified as a significant environmental pollutant. In our work for the Office of Toxic Substances, Versar is studying the economics and other aspects of possible regulatory alternatives as applied to PCB's. We are also studying treatment methods which might be applied to control of waterborne PCB's in industrial effluents. The purpose of this paper is to define current PCB production and use, including process technology and sources of wastes containing PCB's.

Domestic Production and Imports

Monsanto, the sole domestic manufacturer of PCB's, manufactures this chemical in its Sauget, Illinois, plant. The basic raw material is biphenyl, which is manufactured from pure benzene in another Monsanto plant. The PCB manufacturing operation is conducted in two steps. First biphenyl is chlorinated with anhydrous chlorine in presence of ferric chloride to produce crude PCB's and then the crude PCB's are distilled to obtain the finished product. A schematic flow diagram of this process is given in figures 1 and 2.

Monsanto currently produces Aroclors 1221, 1016, 1242, and 1254 (chlorine contents approximately 21, 41, 42, and 54 percent by weight, respectively). For the production of a given product, biphenyl and catalyst are heated to melting and chlorine gas is introduced while the charge is circulated with a pump. The time of chlorine contact controls the degree of product chlorination. Vapors from the chlorination (HCl) are scrubbed and removed to another part of the plant.

The crude product is held at temperature and blown with dry air for several hours, and then is sent to storage where a small amount of lime is added to remove remaining HCl or ferric chloride. The blown air is scrubbed and vented to the atmosphere through a demister.

Purification, as typified by figure 2, varies somewhat between the different products. The 1016 material is processed in a retort and a vacuum distillation (steam jet ejection) to remove the more highly chlorinated compounds. The first cut from the tower is recycled to the retort. When a preset overhead temperature is reached, the product is removed and sent to storage and shipment. The other Aroclors are vacuum distilled (steam jet ejectors), the product being the condensate. The still bottoms (called Montars) are incinerated.

Monsanto's production in 1974 was 40,466,000 lb, of which 34,406,000 was sold domestically and 5,395,000 lb exported. Production has been about at the 40-million-pound level since 1971, and reached a high of 85 million pounds in 1970. Company expectations are for a sizeable increase in sales in 1976, followed by small, steady growth.

Imports in 1973 and 1974 amounted to 1.2 to 1.5 percent of Monsanto's domestic sales in those years. Importation appears to be steady or slightly increasing. The major source of imported PCB's is Italy, from which decachlorobiphenyl made by Caffaro is imported by one company for use in investment casting waxes. This constitutes 80 to 90 percent of the imports; the other 10 to 20 percent originates in France (Prodelec) and is used

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*Vice President, Versar, Inc., Springfield, Virginia.

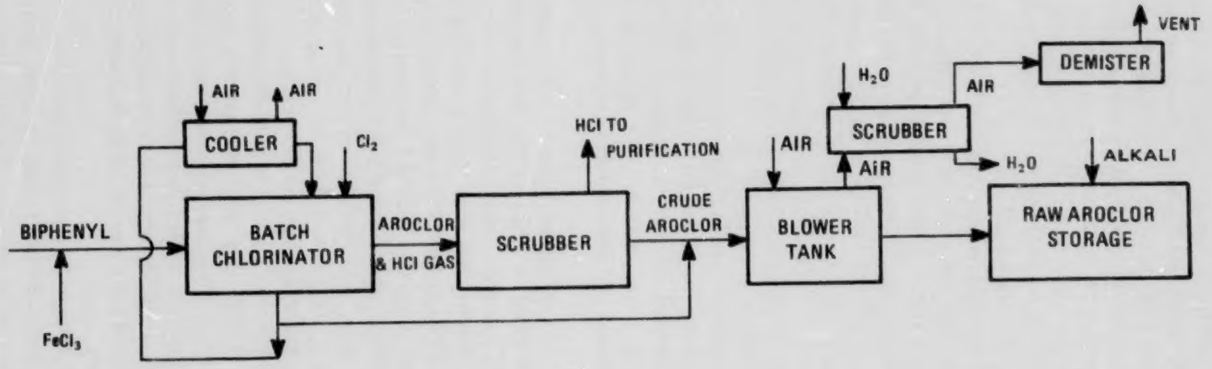

Figure 1. Preparation of crude chlorinated biphenyls Monsanto Krummrich plant.

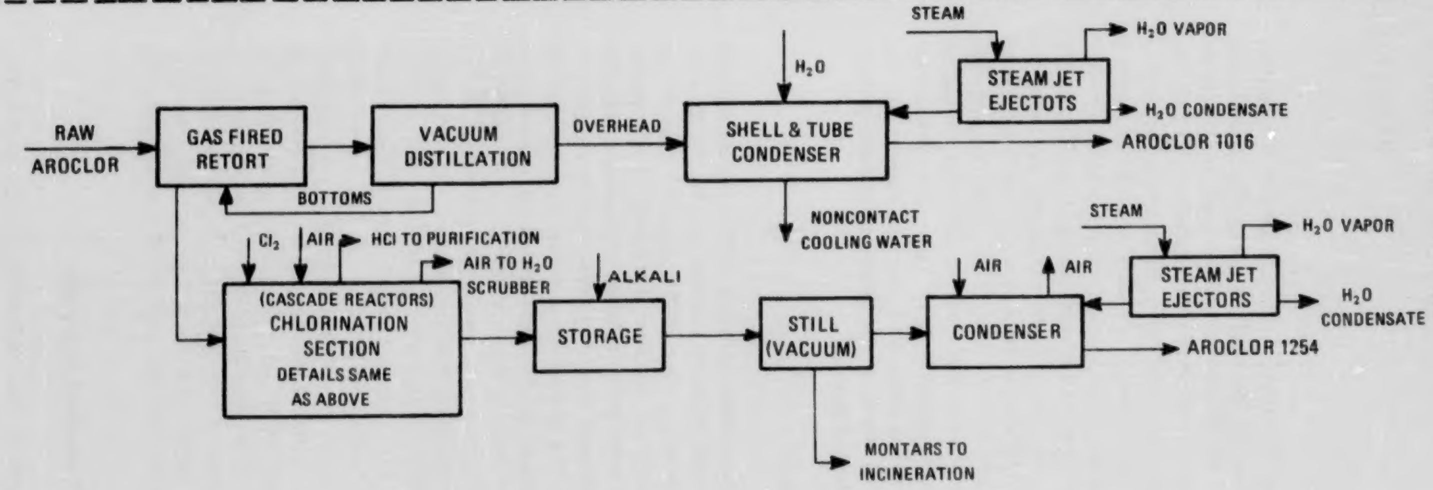

Figure 2. Distillation of crude products Monsanto Krummrich plant.

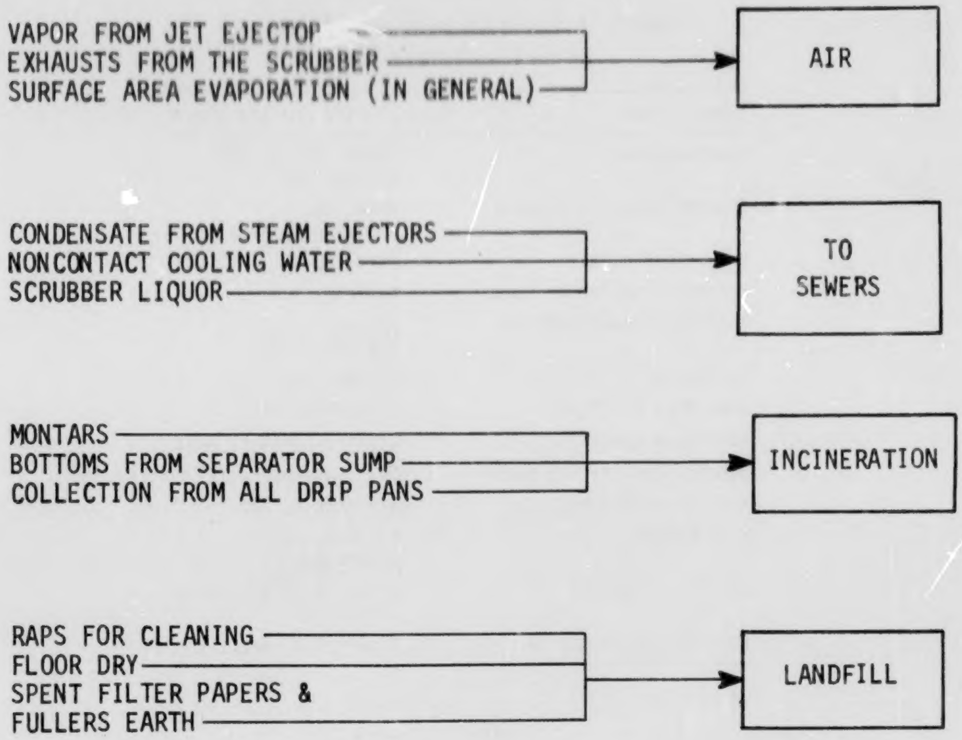
VAPOR FROM JET EJECTOR
EXHAUSTS FROM THE SCRUBBER
SURFACE AREA EVAPORATION (IN GENERAL)
AIR
CONDENSATE FROM STEAM EJECTORS
NONCONTACT COOLING WATER
SCRUBBER LIQUOR
TO SEWERS
MONTARS
BOTTOMS FROM SEPARATOR SUMP
COLLECTION FROM ALL DRIP PANS
INCINERATION
RAPS FOR CLEANING
FLOOR DRY
SPENT FILTER PAPERS & FULLERS EARTH
LANDFILL

Figure 3. Nonproduct PCB discharges at Monsanto's Krummrich plant.

in semiclosed heat transfer applications. Until recently PCB's were used in some U.S. built mining machines, but this use has been discontinued.

In the Monsanto process, PCB's can be released to the environment through the sources listed on figure 3. Waterborne discharges of PCB's at this plant have been reduced greatly over the past few years, and are now below 1 pound per day, according to Monsanto.

Capacitor and Transformer Production

Producers of capacitors and transformers utilize essentially all of Monsanto's domestic sales of PCB's. About 70 percent of the usage is in capacitor production, and, of this total, roughly half is for small capacitors and half for large capacitors. Lists and locations of capacitor and transformer producers are presented in tables 1 and 2, respectively.

Transformers containing PCB's comprise only about 5 percent of the U.S. transformer market, and are used only where safety and reliability are of prime importance. On the other hand, about 95 percent of capacitors made in the United States utilize PCB's. According to General Electric, about 100 X 10^6 capacitors containing PCB's are made in the United States yearly, most for first-time use in products. Life expectancy for capacitors is over 10 years for lighting applications and over 20 years in electric utility service. Life expectancy of transformers containing PCB's is over 30 years, and some 135,000 have been put into service since 1932 (virtually all are still in service).

The steps involving PCB's used in the manufacture of capacitors and transformers include filtration of PCB's, filling (under vacuum), removal of excess PCB's, sealing of units, and heat and electrical testing. Sources of nonproduct discharges of PCB's are roughly similar for capacitor and transformer production plants, and are listed in table 3.

Most of the in-plant PCB wastes which reach water streams originate in the filling area. A brief description of the various filling processes is presented below.

Small capacitor bodies, each containing a machine-

Table 1. U.S. transformer manufacturing industry using PCB's

Company name	Location of the plant
Westinghouse	South Boston, Va. Sharon, Pa.
General Electric Company	Rome, Ga. Pittsfield, Mass.
Research-Cottrell	Finderne, N.J.
Niagara Transformer Corp.	Buffalo, N.Y.
Standard Transformer Co.	Warren, Ohio Medford, Oreg.
Helena Corp.	Helena, Ala.
Hevi-Duty Electric	Goldsboro, N.C.
Kuhlman Electric Co.	Crystal Springs, Miss.
Electro Engineering Works	San Leandro, Calif.
R.E. Uptegraff Mfg. Co.	Scottsdale, Pa.
H.K. Porter	Belmont, Calif. Lynchburg, Va.
Van Tran Electric Co.	Vandalia, Ill. Waco, Tex.
Esco Manufacturing Co.	Greenville, Tex.

Table 2. U.S. capacitor manufacturing industry using PCB's

Company name (In order of PCB's usage)	Location of the plant
General Electric Company	Hudson Falls, N.Y. Ft. Edward, N.Y.
Aerovox	New Bedford, Mass.
Universal Manufacturing Corp.	Bridgeport, Conn. Totowa, N.J.
Westinghouse Electric Corp.	Bloomington, Ind.
Cornell Dubilier	New Bedford, Mass.
P.R. Mallory & Co., Inc.	Waynesboro, Tenn.
Sangamo Electric Co.	Pickens, S.C.
Sprague Electric Co.	North Adams, Mass.
Electric Utility Co.	LaSalle, Ill.
Capacitor Specialists, Inc.	Escondido, Calif.
JARD Corp.	Bennington, Vt.
York Electronics	Brooklyn, N.Y.
McGraw-Edison	Greenwood, S.C.
RF Interonics	Bayshore, L.I., N.Y.
Axel Electronic, Inc.	Jamaica, N.Y.
Tobe Deutschmann Labs.	Canton, Mass.
Cine-Chrome Lab, Inc.	Palo Alto, Calif.

Table 3. Sources of nonproduct discharges of PCB's

Source	Type of effluent/ typical disposal
Vacuum pumps	Air
Evaporation and ventilation	Air
Transfer spillage	Water
Vacuum pump cooling water or condensate (steam jet)	Water
Personal hygiene	Water (Municipal Sewer)
Reject products	Landfill
Floor and equipment cleanup wastes	Landfill
Drip pan collection	Incineration
Filter media	Landfill

wrapped helix of alternating paper and aluminum foil, are placed in a chamber, the chamber is evacuated, and prefiltered dielectric oil is introduced until it covers the capacitors. The vacuum assures that the paper will be well soaked (impregnated) with the fluid.

After the filling operation the vacuum is released, excess fluid is removed to a holding tank, and the capacitors are sealed and then deoiled. Then they undergo heating electric tests and, if specifications are met, are packaged. Reject capacitors and, in many cases, solid wastes containing PCB's are landfilled.

Large capacitors are produced similarly, although some differences can be observed:

1. Large capacitors (over 2 to 3 lb of PCB's) are typically filled through a small fill hole, while the fill tank is under vacuum. Large capacitors may be filled separately or flood-filled in a chamber. Some plants use a multicell "carousel" arangement where loading and unloading occurs at one station, and the capacitor(s) in each cell are progressively dried, evacuated, filled, and the fluid drained from the cell at stations along the carousel.
2. Following the filling operation, the fill holes in the large capacitors are sealed or plugged with solder, deoiled, and then tested prior to packaging and shipment.

Transformer filling is very similar to the filling of the larger capacitors, but transformers are all filled individually. Individual filling appears to generate less waste PCB's from spills, drip, and deoiling operations. If a transformer does not meet test specifications, the fluid is drained, filtered, and returned to the supply tank, and the windings assembly and other parts are inspected to identify the cause. Once repaired, the transformer reenters the assembly line.

Investment Casting Process

Imported decachlorobiphenyl (deka) is used as a filler for investment casting waxes; the estimated yearly usage is about 400,000 lb, some of which is in exported waxes. In investment casting, a pattern is made of the wax and a ceramic mold is formed on the wax. The wax is melted out, and the mold, after baking out in a furnace, is ready for use. The wax used to form the pattern contains about 60 percent used wax and 40 percent virgin pattern wax. Significant losses of PCB's to the atmosphere can occur in the melting out process and in the furnace. Used wax can possibly be reclaimed, but most appears to be landfilled. Polychlorinated triphenyls are also used as pattern wax fillers.

PCB DISPOSAL, RECLAIMING, AND TREATMENT

Thomas E. Kopp*

Abstract

The Environmental Protection Agency in 1975 sought information about the uses, disposal, and effects of PCB's from 84 purchasers and importers of PCB's. Responses indicate that while PCB's were formerly disposed of in landfills, high-temperature incineration is now prevalent. There is no proven technology for removal of PCB's from water, and emphasis is on preventing production-line PCB's from entering waste water. EPA is presently formulating criteria for ambient PCB levels in water and is developing a new toxic effluent standard.

On August 16, 1975, the Office of Enforcement, EPA, sent out 84 letters (figure 1) to companies in the United States that purchase PCB's from Monsanto, to those that import them, and to others that may be users. This letter has been modified to include polychlorinated terphenyls, PCT's, both because of their similar structure and because they are a type of chemical that we will have evaluated in the whole program of PCB's. This letter concluded with four pages of questions which covered the uses, importation, reclaiming, disposal of PCB's (figure 2) and which concerned effluents, transportation, spills, health and environmental effects, and other data the companies had on file (figure 3).

Since then, we have received 83 responses and we are in the process of evaluating and assembling the information into a useable form for making decisions within the Federal Government. This information will be made available to the public and to local and State governments. This information will be handled in accordance with the confidentiality requested by the companies and the Trade Secrets Act.

Starting in 1972, industry had developed a standard for handling and disposal of PCB's (figure 4). In 1974, this standard was finalized and published. It covers the following points (figure 5): housekeeping, disposal, effluents, analytical methods, and several other important areas. This is a voluntary standard for industry and for the Federal Government. We encourage the implementation of this standard support by everybody and also support the proposal by the National Electrical Manufacturers Association to revise and update it in the near future.

Disposal of PCB's has historically been to the local landfill site or to a scrap oil dealer. Since the concern with the environmental problems associated with PCB's has become evident, industry has developed several ways of disposing of its PCB waste (figure 6). High-temperature incineration is the most effective way we know today because it will completely destroy PCB's. The other methods are only to contain the material and not to let PCB's get out into the environment.

In Canada, there is a study going on about which we have little knowledge (figure 7). It appears to be a method by which PCB's may be reclaimed after their useful lives.

On Thursday, December 27, 1973, EPA issued its proposed toxic pollutant effluent standards under Section 307a of the Water Act. During the hearings in May, 1974, effluent treatment was discussed (figure 8). Westinghouse stated that "A review of techniques for removal of materials such as PCB's from the water has revealed that no commercial operation is now in effect. Furthermore, there is no proven technology on which such a technique might be based." General Electric stated that "The control of PCB's from current production is basically a containment process. Water is not an essential component of the manufacturing process, so the problem is not one of removing a contaminant from an aqueous process stream, but of preventing the adventitious loss of PCB's from the production line to a waste water line." Monsanto stated for its production facility that "the bathtub approach, which really involves paving, curbing, isolating the drains, ... etc.," is the only known method for treatment.

We are basically at the same place today as we were in May of 1974. As EPA develops its reproposed toxic effluent standard, which should be published the first half of next year, we will be looking at carbon absorbtion, reverse osmosis, and ozonation.

EPA is almost ready to send out for interagency and State review of the Criteria for Water Quality, Section 304a of the Water Act (figure 9). This is not a rulemaking procedure so it does not have to go through hearings and rulings. This is the ambient level which we desire will not be exceeded in the Nation's waters.

There have been 20 spills identified at this time which have involved PCB's (figure 10). Only six of these were reported to EPA, and only over these did we have any control during the cleanup. There is only one out of

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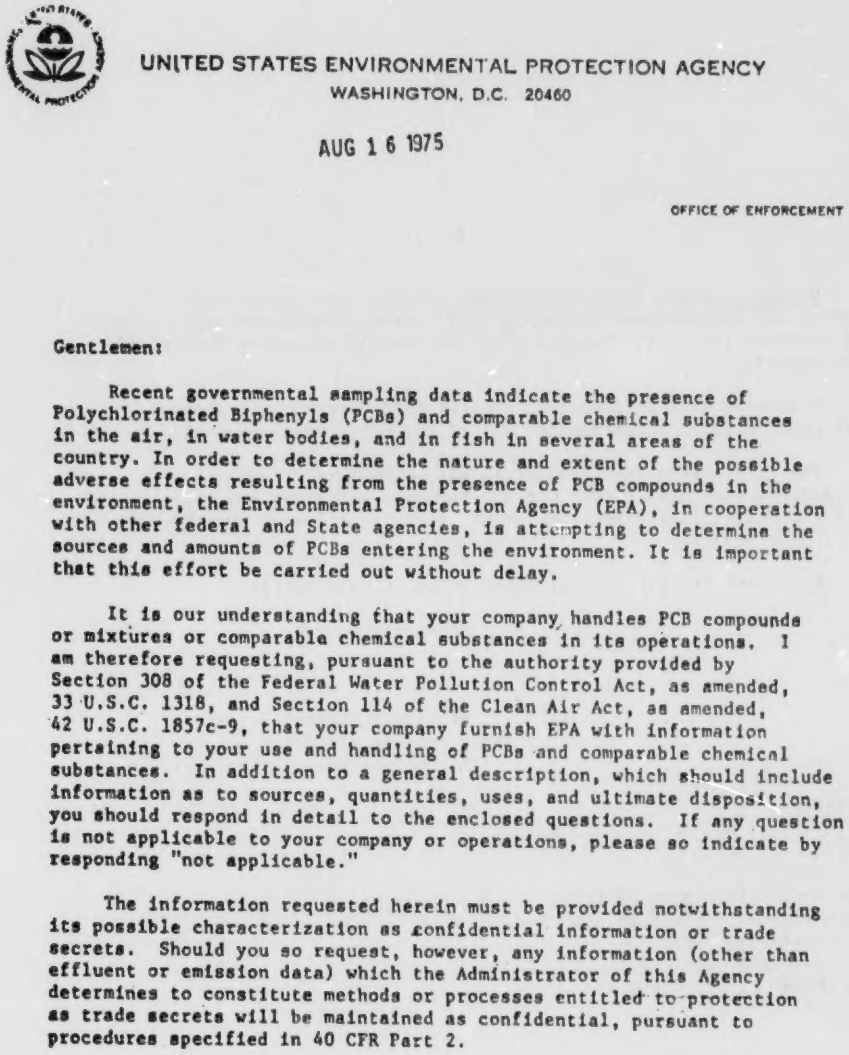
*Environmental Protection Agency, Office of Toxic Substances (WH-557), Washington, D.C.



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

AUG 1 6 1975

OFFICE OF ENFORCEMENT

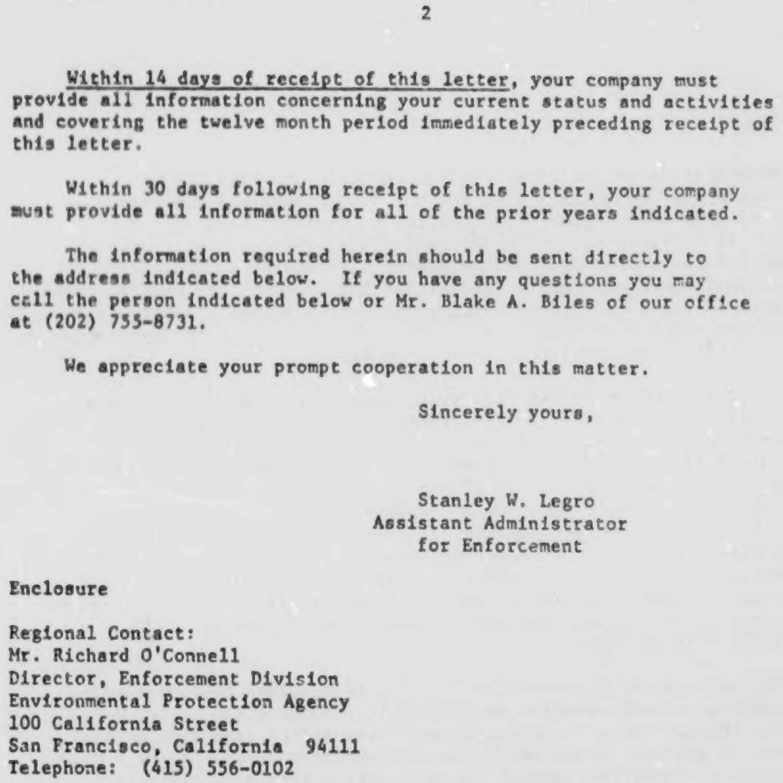
Gentlemen:

Recent governmental sampling data indicate the presence of Polychlorinated Biphenyls (PCBs) and comparable chemical substances in the air, in water bodies, and in fish in several areas of the country. In order to determine the nature and extent of the possible adverse effects resulting from the presence of PCB compounds in the environment, the Environmental Protection Agency (EPA), in cooperation with other federal and State agencies, is attempting to determine the sources and amounts of PCBs entering the environment. It is important that this effort be carried out without delay.

It is our understanding that your company handles PCB compounds or mixtures or comparable chemical substances in its operations. I am therefore requesting, pursuant to the authority provided by Section 308 of the Federal Water Pollution Control Act, as amended, 33 U.S.C. 1318, and Section 114 of the Clean Air Act, as amended, 42 U.S.C. 1857c-9, that your company furnish EPA with information pertaining to your use and handling of PCBs and comparable chemical substances. In addition to a general description, which should include information as to sources, quantities, uses, and ultimate disposition, you should respond in detail to the enclosed questions. If any question is not applicable to your company or operations, please so indicate by responding "not applicable."

The information requested herein must be provided notwithstanding its possible characterization as confidential information or trade secrets. Should you so request, however, any information (other than effluent or emission data) which the Administrator of this Agency determines to constitute methods or processes entitled to protection as trade secrets will be maintained as confidential, pursuant to procedures specified in 40 CFR Part 2.

Figure 1. Letter from EPA to purchasers, importers, and users of PCB's.



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Within 14 days of receipt of this letter, your company must provide all information concerning your current status and activities and covering the twelve month period immediately preceding receipt of this letter.

Within 30 days following receipt of this letter, your company must provide all information for all of the prior years indicated.

The information required herein should be sent directly to the address indicated below. If you have any questions you may call the person indicated below or Mr. Blake A. Biles of our office at (202) 755-8731.

We appreciate your prompt cooperation in this matter.

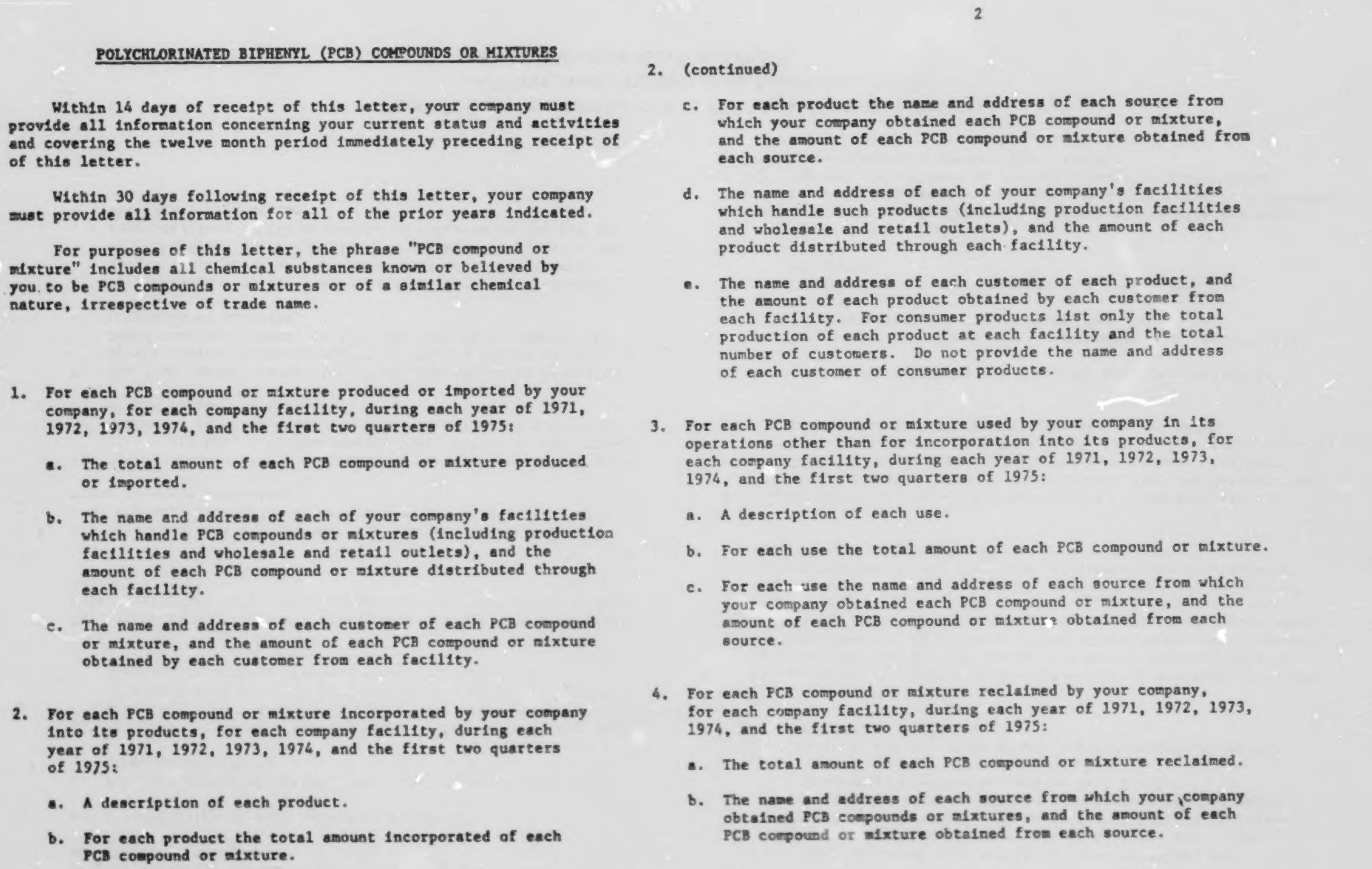
Sincerely yours,

Stanley W. Legro
Assistant Administrator
for Enforcement

Enclosure

Regional Contact:
Mr. Richard O'Connell
Director, Enforcement Division
Environmental Protection Agency
100 California Street
San Francisco, California 94111
Telephone: (415) 556-0102

Figure 1. Letter from EPA to purchasers, importers, and users of PCB's (con.).



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POLYCHLORINATED BIPHENYL (PCB) COMPOUNDS OR MIXTURES

Within 14 days of receipt of this letter, your company must provide all information concerning your current status and activities and covering the twelve month period immediately preceding receipt of of this letter.

Within 30 days following receipt of this letter, your company must provide all information for all of the prior years indicated.

For purposes of this letter, the phrase "PCB compound or mixture" includes all chemical substances known or believed by you to be PCB compounds or mixtures or of a similar chemical nature, irrespective of trade name.

1. For each PCB compound or mixture produced or imported by your company, for each company facility, during each year of 1971, 1972, 1973, 1974, and the first two quarters of 1975:

   a. The total amount of each PCB compound or mixture produced or imported.

   b. The name and address of each of your company's facilities which handle PCB compounds or mixtures (including production facilities and wholesale and retail outlets), and the amount of each PCB compound or mixture distributed through each facility.

   c. The name and address of each customer of each PCB compound or mixture, and the amount of each PCB compound or mixture obtained by each customer from each facility.

2. For each PCB compound or mixture incorporated by your company into its products, for each company facility, during each year of 1971, 1972, 1973, 1974, and the first two quarters of 1975:

   a. A description of each product.

   b. For each product the total amount incorporated of each PCB compound or mixture.

2. (continued)

   c. For each product the name and address of each source from which your company obtained each PCB compound or mixture, and the amount of each PCB compound or mixture obtained from each source.

   d. The name and address of each of your company's facilities which handle such products (including production facilities and wholesale and retail outlets), and the amount of each product distributed through each facility.

   e. The name and address of each customer of each product, and the amount of each product obtained by each customer from each facility. For consumer products list only the total production of each product at each facility and the total number of customers. Do not provide the name and address of each customer of consumer products.

3. For each PCB compound or mixture used by your company in its operations other than for incorporation into its products, for each company facility, during each year of 1971, 1972, 1973, 1974, and the first two quarters of 1975:

   a. A description of each use.

   b. For each use the total amount of each PCB compound or mixture.

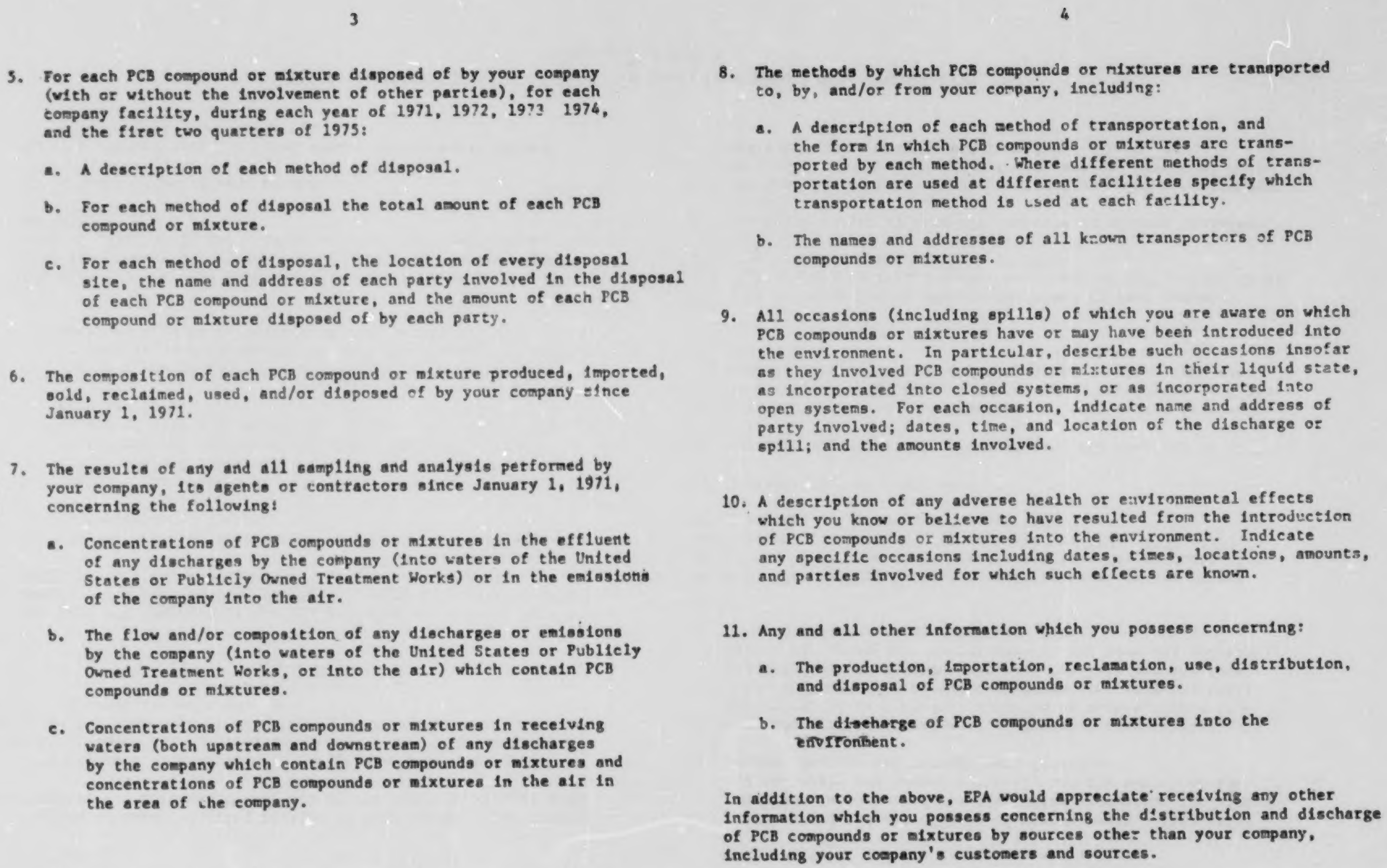
   c. For each use the name and address of each source from which your company obtained each PCB compound or mixture, and the amount of each PCB compound or mixture obtained from each source.

4. For each PCB compound or mixture reclaimed by your company, for each company facility, during each year of 1971, 1972, 1973, 1974, and the first two quarters of 1975:

   a. The total amount of each PCB compound or mixture reclaimed.

   b. The name and address of each source from which your company obtained PCB compounds or mixtures, and the amount of each PCB compound or mixture obtained from each source.

Figure 2. Questions sent by EPA to PCB users, concerning uses, importation, and reclaiming of PCB's.



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5. For each PCB compound or mixture disposed of by your company (with or without the involvement of other parties), for each company facility, during each year of 1971, 1972, 1973 1974, and the first two quarters of 1975:

   a. A description of each method of disposal.

   b. For each method of disposal the total amount of each PCB compound or mixture.

   c. For each method of disposal, the location of every disposal site, the name and address of each party involved in the disposal of each PCB compound or mixture, and the amount of each PCB compound or mixture disposed of by each party.

6. The composition of each PCB compound or mixture produced, imported, sold, reclaimed, used, and/or disposed of by your company since January 1, 1971.

7. The results of any and all sampling and analysis performed by your company, its agents or contractors since January 1, 1971, concerning the following:

   a. Concentrations of PCB compounds or mixtures in the effluent of any discharges by the company (into waters of the United States or Publicly Owned Treatment Works) or in the emissions of the company into the air.

   b. The flow and/or composition of any discharges or emissions by the company (into waters of the United States or Publicly Owned Treatment Works, or into the air) which contain PCB compounds or mixtures.

   c. Concentrations of PCB compounds or mixtures in receiving waters (both upstream and downstream) of any discharges by the company which contain PCB compounds or mixtures and concentrations of PCB compounds or mixtures in the air in the area of the company.

4

8. The methods by which PCB compounds or mixtures are transported to, by, and/or from your company, including:

   a. A description of each method of transportation, and the form in which PCB compounds or mixtures are transported by each method. Where different methods of transportation are used at different facilities specify which transportation method is used at each facility.

   b. The names and addresses of all known transporters of PCB compounds or mixtures.

9. All occasions (including spills) of which you are aware on which PCB compounds or mixtures have or may have been introduced into the environment. In particular, describe such occasions insofar as they involved PCB compounds or mixtures in their liquid state, as incorporated into closed systems, or as incorporated into open systems. For each occasion, indicate name and address of party involved; dates, time, and location of the discharge or spill; and the amounts involved.

10. A description of any adverse health or environmental effects which you know or believe to have resulted from the introduction of PCB compounds or mixtures into the environment. Indicate any specific occasions including dates, times, locations, amounts, and parties involved for which such effects are known.

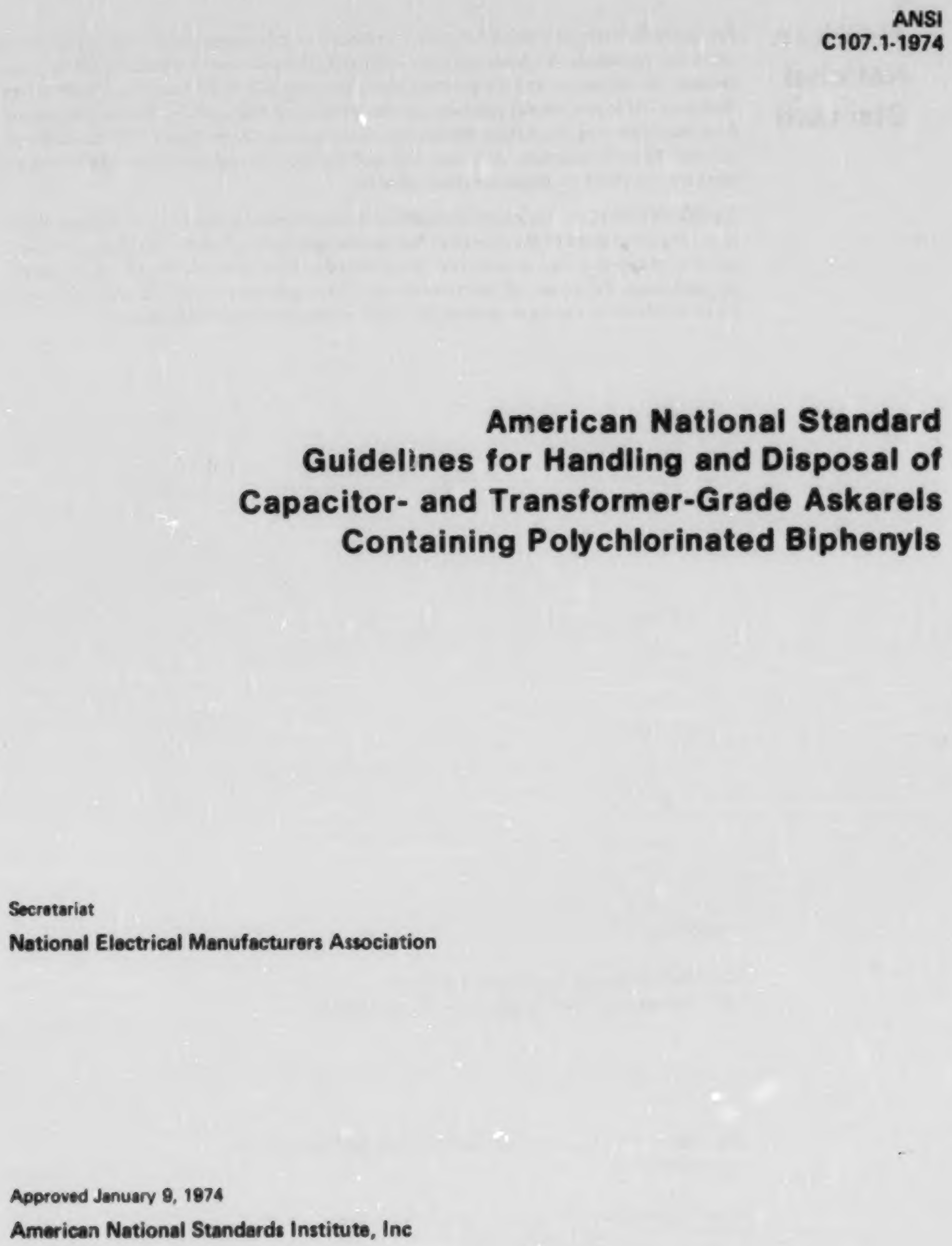
11. Any and all other information which you possess concerning:

   a. The production, importation, reclamation, use, distribution, and disposal of PCB compounds or mixtures.

   b. The discharge of PCB compounds or mixtures into the environment.

In addition to the above, EPA would appreciate receiving any other information which you possess concerning the distribution and discharge of PCB compounds or mixtures by sources other than your company, including your company's customers and sources.

Figure 3. Questions sent by EPA to PCB users, concerning disposal, effluents, transportation, spills, hearth and environmental effects, and other data about PCB's.



ANSI
C107.1-1974

American National Standard
Guidelines for Handling and Disposal of
Capacitor- and Transformer-Grade Askarels
Containing Polychlorinated Biphenyls

Secretariat
National Electrical Manufacturers Association

Approved January 9, 1974
American National Standards Institute, Inc

Figure 4. Cover of industry standard for handling and disposal of PCB's.

American National Standard

An American National Standard implies a consensus of those substantially concerned with its scope and provisions. An American National Standard is intended as a guide to aid the manufacturer, the consumer, and the general public. The existence of an American National Standard does not in any respect preclude anyone, whether he has approved the standard or not, from manufacturing, marketing, purchasing, or using products, processes, or procedures not conforming to the standard. American National Standards are subject to periodic review and users are cautioned to obtain the latest editions.

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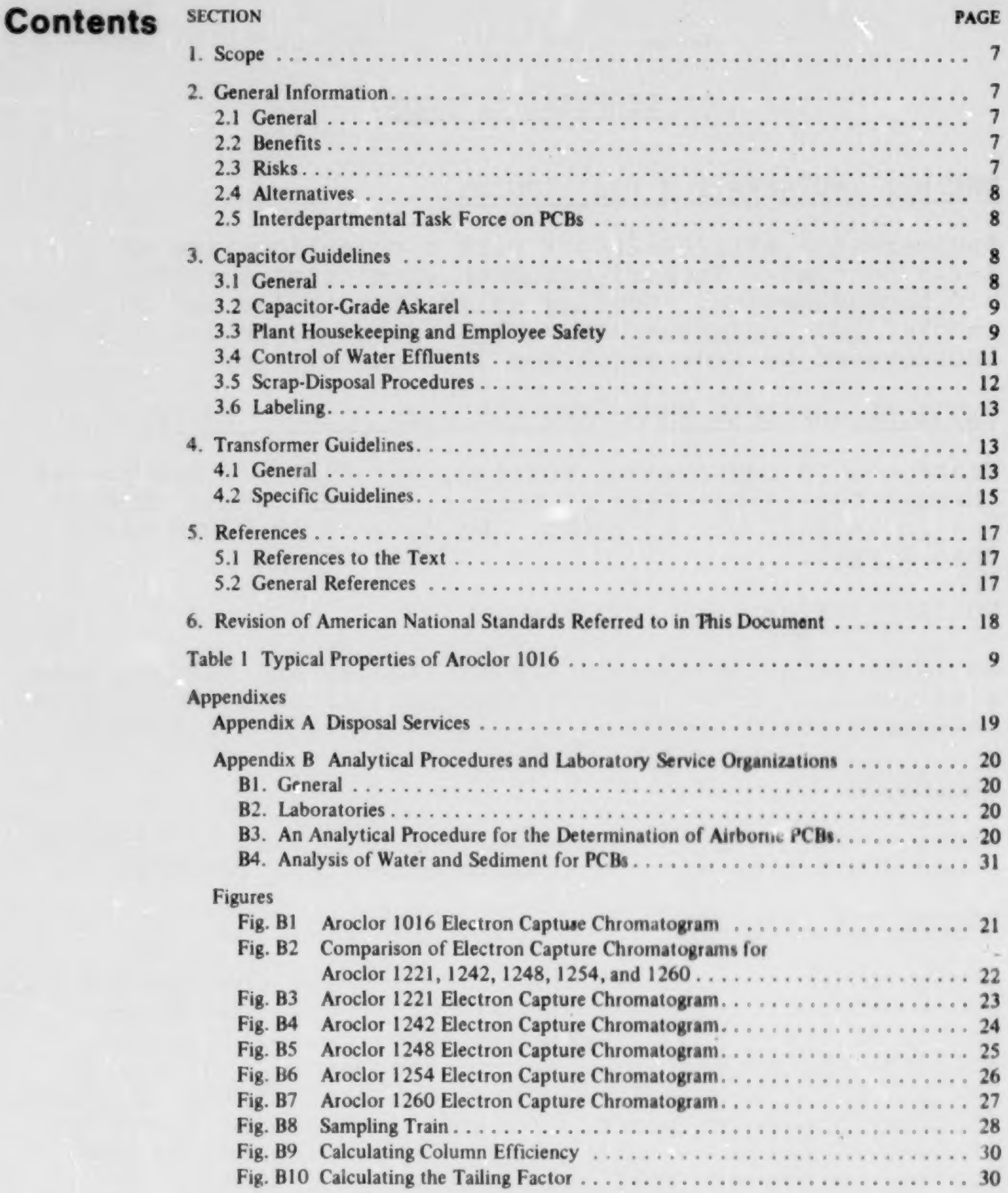

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Figure 5. Points covered in industry guideline for handling and disposal of PCB's.

DISPOSAL METHODS

THERMAL INCINERATION FOR LIQUIDS

Incineration at 2200-2600°F with a retention time of 1-1.5 seconds. Venturi scrubber eliminates possibility air contamination. Cooling water is recycled and routinely checked for contamination. High aqeous waste are also incinerated by side injection into the system.

PHYSICAL CHEMICAL TREATMENT FOR HIGH AQUEOUS WASTE

Waste are pH controlled, settled, and filtered and passed through two carbon beds in series. Water passed through has invariably met at specs. No detectable PCB's (less than 2 ppb).

SOLIDIFICATION

Solidification means solidifying liquids and sludges using a silicates-cement powder technique. After solidification, materials are disposed of in lined scientific landfill.

GELLATION

Liquids and sludges are polymerized or gelled in steel of fiber drumswhich are placed in a scientific landfill

SCIENTIFIC LANDFILL

Series of reinforced hypalon or reinforced chlorinated polyethylene lined cells in clay oil areas into which solid, sludges, stabilized chemicals, gelled liquids, solidfill liquids or sludges are disposited. A sump at the bottom of each cell recycles leachate to the physical-chemical treatment plant. A three-dimensional inventory is kept of waste in each cell. Surrounding ground and surface waters are routinely checked each month.

Figure 6. Methods developed by industry for disposal of PCB waste.

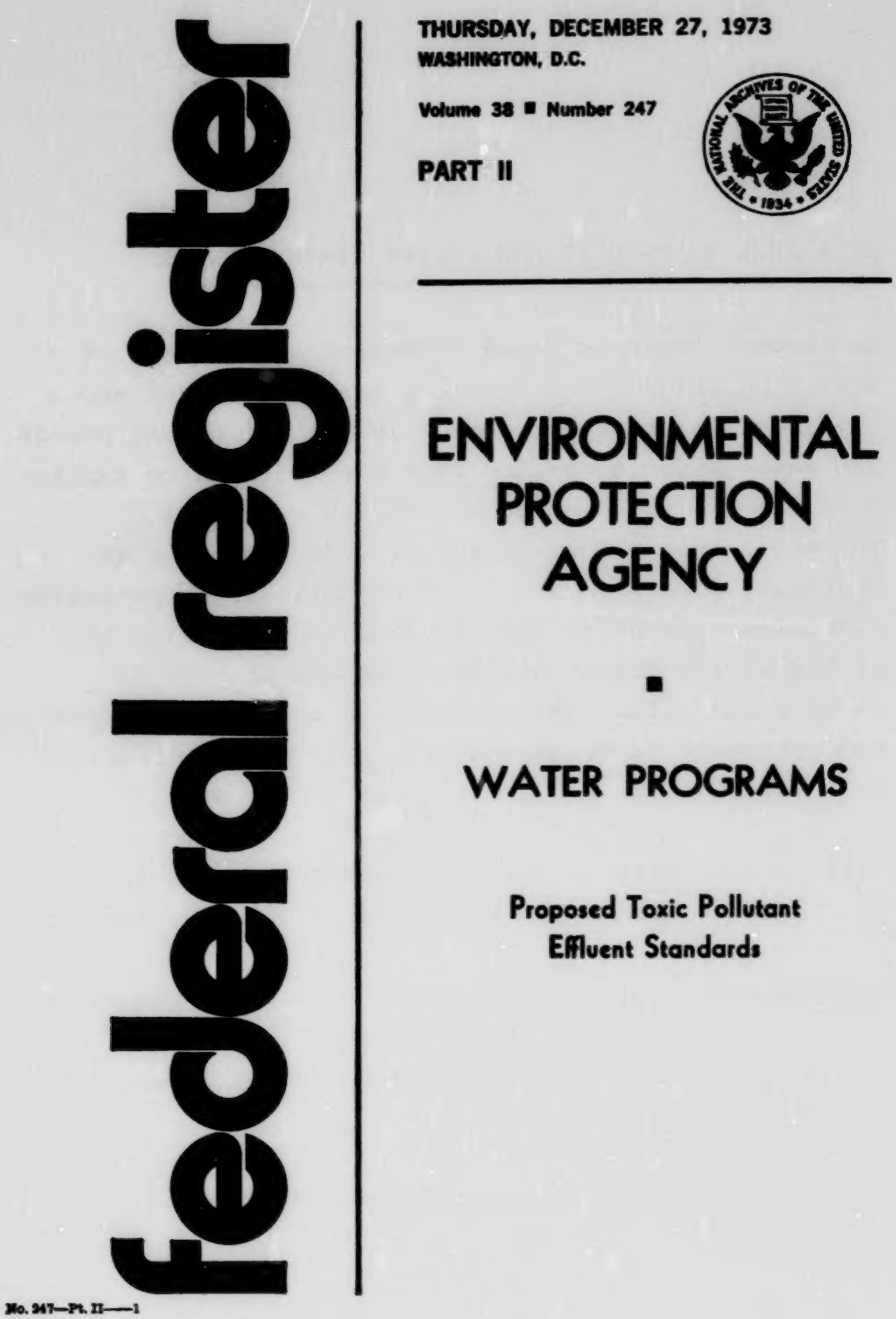
AS A FUEL TO PRODUCE LOW ALKALI CEMENT POWDER

In Canada, there is being pioneered the concept of utilizing chlorinated chemical waste as a fuel and a supply of chlorine to produce low alkali cement powder. The waste material is fed into the fire box to supplement the fuel oil and burns at 2200 - 2600° f. The waste breaks down to water, carbon dioxide and hydrochloric acid. The acid neutralizes the potassium hydroxide present in the raw feed thus reducing the alkali in the cement powder. The cement kiln is perhaps the safest way to destroy PCBs. The temperature and retention time, approximately 16 - 18 seconds, are sufficient to reduce the PCBs.

This test program is currently under way at the St. Lawrence Cement Co. in Hamilton, Ontario, Canada.

Participants are: Ontario Water Resources Commission
St. Lawrence Cement Company
Chem-Trol Pollution Services, Inc.

Figure 7. Description of Canadian PCB study.



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THE NATIONAL ARCHIVES OF THE UNITED STATES
1934

ENVIRONMENTAL
PROTECTION
AGENCY

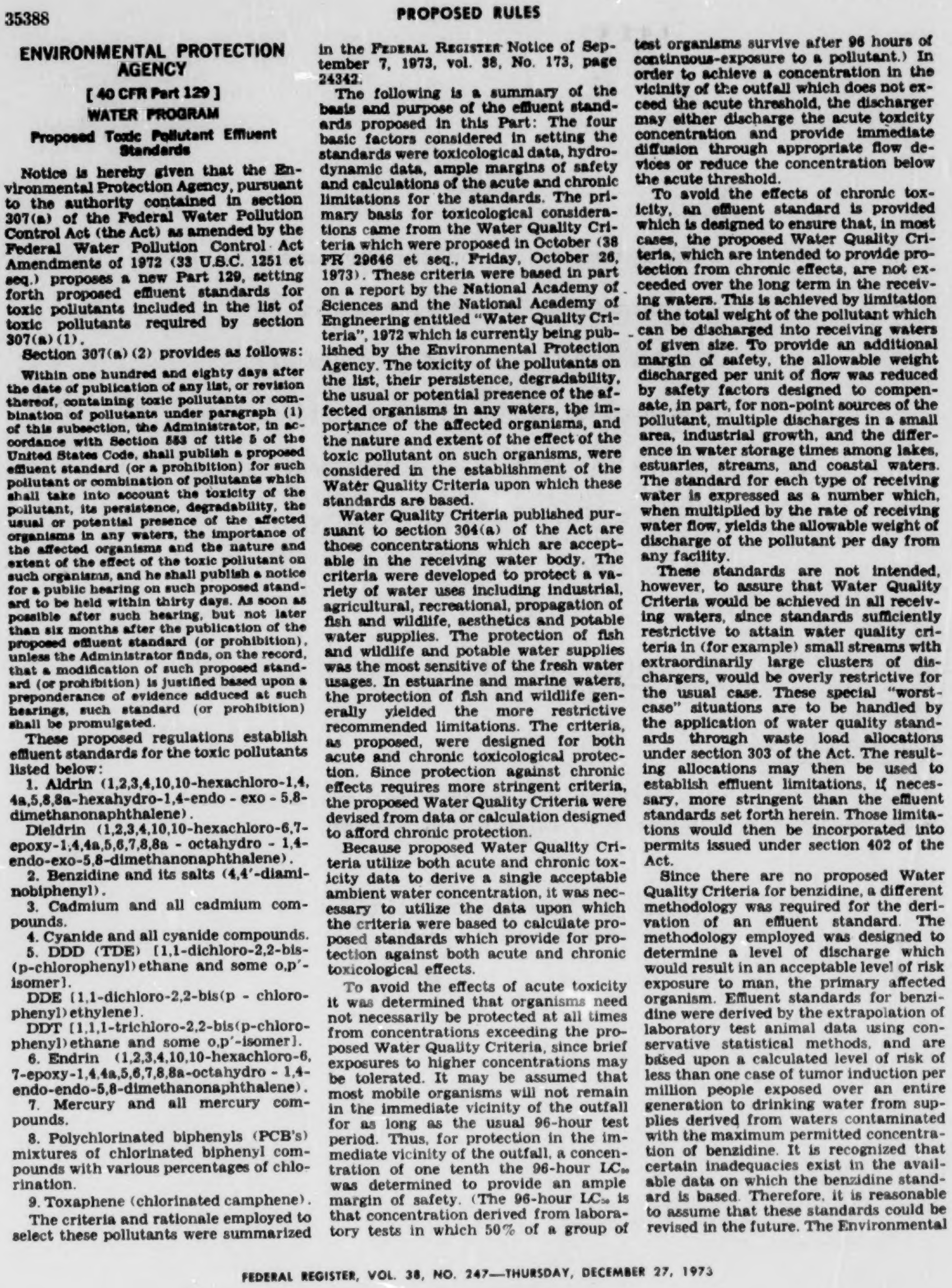
■

WATER PROGRAMS

Proposed Toxic Pollutant
Effluent Standards

No. 247—Pt. II——1

Figure 8. EPA proposed toxic pollutant effluent standards.



ENVIRONMENTAL PROTECTION AGENCY

[40 CFR Part 129]

WATER PROGRAM

Proposed Toxic Pollutant Effluent Standards

Notice is hereby given that the Environmental Protection Agency, pursuant to the authority contained in section 307(a) of the Federal Water Pollution Control Act (the Act) as amended by the Federal Water Pollution Control Act Amendments of 1972 (33 U.S.C. 1251 et seq.) proposes a new Part 129, setting forth proposed effluent standards for toxic pollutants included in the list of toxic pollutants required by section 307(a)(1).

Section 307(a)(2) provides as follows:

> Within one hundred and eighty days after the date of publication of any list, or revision thereof, containing toxic pollutants or combination of pollutants under paragraph (1) of this subsection, the Administrator, in accordance with Section 553 of title 5 of the United States Code, shall publish a proposed effluent standard (or a prohibition) for such pollutant or combination of pollutants which shall take into account the toxicity of the pollutant, its persistence, degradability, the usual or potential presence of the affected organisms in any waters, the importance of the affected organisms and the nature and extent of the effect of the toxic pollutant on such organisms, and he shall publish a notice for a public hearing on such proposed standard to be held within thirty days. As soon as possible after such hearing, but not later than six months after the publication of the proposed effluent standard (or prohibition), unless the Administrator finds, on the record, that a modification of such proposed standard (or prohibition) is justified based upon a preponderance of evidence adduced at such hearings, such standard (or prohibition) shall be promulgated.

These proposed regulations establish effluent standards for the toxic pollutants listed below:

1. Aldrin (1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4-endo - exo - 5,8-dimethanonaphthalene).

Dieldrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a - octahydro - 1,4-endo-exo-5,8-dimethanonaphthalene).

2. Benzidine and its salts (4,4′-diaminobiphenyl).

3. Cadmium and all cadmium compounds.

4. Cyanide and all cyanide compounds.

5. DDD (TDE) [1,1-dichloro-2,2-bis-(p-chlorophenyl)ethane and some o,p′-isomer].

DDE [1,1-dichloro-2,2-bis(p - chlorophenyl)ethylene].

DDT [1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane and some o,p′-isomer].

6. Endrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro - 1,4-endo-endo-5,8-dimethanonaphthalene).

7. Mercury and all mercury compounds.

8. Polychlorinated biphenyls (PCB's) mixtures of chlorinated biphenyl compounds with various percentages of chlorination.

9. Toxaphene (chlorinated camphene).

The criteria and rationale employed to select these pollutants were summarized in the FEDERAL REGISTER Notice of September 7, 1973, vol. 38, No. 173, page 24342.

The following is a summary of the basis and purpose of the effluent standards proposed in this Part: The four basic factors considered in setting the standards were toxicological data, hydrodynamic data, ample margins of safety and calculations of the acute and chronic limitations for the standards. The primary basis for toxicological considerations came from the Water Quality Criteria which were proposed in October (38 FR 29646 et seq., Friday, October 26, 1973). These criteria were based in part on a report by the National Academy of Sciences and the National Academy of Engineering entitled "Water Quality Criteria", 1972 which is currently being published by the Environmental Protection Agency. The toxicity of the pollutants on the list, their persistence, degradability, the usual or potential presence of the affected organisms in any waters, the importance of the affected organisms, and the nature and extent of the effect of the toxic pollutant on such organisms, were considered in the establishment of the Water Quality Criteria upon which these standards are based.

Water Quality Criteria published pursuant to section 304(a) of the Act are those concentrations which are acceptable in the receiving water body. The criteria were developed to protect a variety of water uses including industrial, agricultural, recreational, propagation of fish and wildlife, aesthetics and potable water supplies. The protection of fish and wildlife and potable water supplies was the most sensitive of the fresh water usages. In estuarine and marine waters, the protection of fish and wildlife generally yielded the more restrictive recommended limitations. The criteria, as proposed, were designed for both acute and chronic toxicological protection. Since protection against chronic effects requires more stringent criteria, the proposed Water Quality Criteria were devised from data or calculation designed to afford chronic protection.

Because proposed Water Quality Criteria utilize both acute and chronic toxicity data to derive a single acceptable ambient water concentration, it was necessary to utilize the data upon which the criteria were based to calculate proposed standards which provide for protection against both acute and chronic toxicological effects.

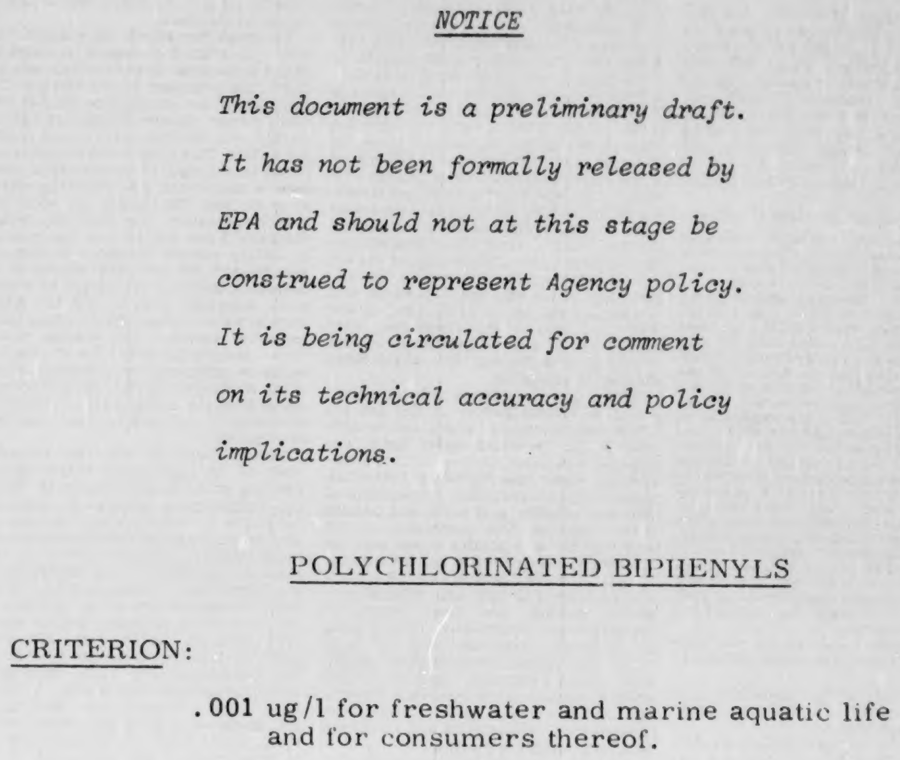
To avoid the effects of acute toxicity it was determined that organisms need not necessarily be protected at all times from concentrations exceeding the proposed Water Quality Criteria, since brief exposures to higher concentrations may be tolerated. It may be assumed that most mobile organisms will not remain in the immediate vicinity of the outfall for as long as the usual 96-hour test period. Thus, for protection in the immediate vicinity of the outfall, a concentration of one tenth the 96-hour LC_{50} was determined to provide an ample margin of safety. (The 96-hour LC_{50} is that concentration derived from laboratory tests in which 50% of a group of test organisms survive after 96 hours of continuous-exposure to a pollutant.) In order to achieve a concentration in the vicinity of the outfall which does not exceed the acute threshold, the discharger may either discharge the acute toxicity concentration and provide immediate diffusion through appropriate flow devices or reduce the concentration below the acute threshold.

To avoid the effects of chronic toxicity, an effluent standard is provided which is designed to ensure that, in most cases, the proposed Water Quality Criteria, which are intended to provide protection from chronic effects, are not exceeded over the long term in the receiving waters. This is achieved by limitation of the total weight of the pollutant which can be discharged into receiving waters of given size. To provide an additional margin of safety, the allowable weight discharged per unit of flow was reduced by safety factors designed to compensate, in part, for non-point sources of the pollutant, multiple discharges in a small area, industrial growth, and the difference in water storage times among lakes, estuaries, streams, and coastal waters. The standard for each type of receiving water is expressed as a number which, when multiplied by the rate of receiving water flow, yields the allowable weight of discharge of the pollutant per day from any facility.

These standards are not intended, however, to assure that Water Quality Criteria would be achieved in all receiving waters, since standards sufficiently restrictive to attain water quality criteria in (for example) small streams with extraordinarily large clusters of dischargers, would be overly restrictive for the usual case. These special "worst-case" situations are to be handled by the application of water quality standards through waste load allocations under section 303 of the Act. The resulting allocations may then be used to establish effluent limitations, if necessary, more stringent than the effluent standards set forth herein. Those limitations would then be incorporated into permits issued under section 402 of the Act.

Since there are no proposed Water Quality Criteria for benzidine, a different methodology was required for the derivation of an effluent standard. The methodology employed was designed to determine a level of discharge which would result in an acceptable level of risk exposure to man, the primary affected organism. Effluent standards for benzidine were derived by the extrapolation of laboratory test animal data using conservative statistical methods, and are based upon a calculated level of risk of less than one case of tumor induction per million people exposed over an entire generation to drinking water from supplies derived from waters contaminated with the maximum permitted concentration of benzidine. It is recognized that certain inadequacies exist in the available data on which the benzidine standard is based. Therefore, it is reasonable to assume that these standards could be revised in the future. The Environmental

Figure 8. (con.).



NOTICE

This document is a preliminary draft. It has not been formally released by EPA and should not at this stage be construed to represent Agency policy. It is being circulated for comment on its technical accuracy and policy implications.

POLYCHLORINATED BIPHENYLS

CRITERION:

.001 ug/l for freshwater and marine aquatic life and for consumers thereof.

Figure 9. Preliminary draft of EPA standard for water quality.

DATE	CIRCUMSTANCES	CITY STATE	AMOUNT SPILLED	AMOUNT RECOVERED	METHOD OF DISPOSAL	COST	REPORTED TO EPA
6/ 9/71	Drums containing 8,600 lb started to leak en route	En route	Unknown	Unknown	None		No
10/12/71	Drum leaking en route, sent back	En route	Unknown	None	None		No
3/72	Rail car develops a leak	En route	Unknown	Unknown	Unknown		No
3/ 8/72	Truck accident, 5 transformers	Roanoke Rapids, NC	1000 gals Inerteen	Unknown	Unknown		No
3/ 8/73	Truck en route developed leak	Kingston, Tenn.	630 gal PCBs	11,500 drums of contaminated soil	55 gal sealed in concrete	$1.7 million	Yes
8/ 9/73	Truck accident	Havana, Ill.	400 gal	Unknown	Unknown		Yes
9/73	Transformer leak	Anderson, Ind.	15 gal	Unknown	Unknown		No
1/ 6/74	Transformer leak	Tuscaloosa Ala.	80-100 gal	Unknown	Unknown		No
4/74	Transformer leak	Louisburg, Kansas	60 gal	Unknown	Unknown		No
5/20/74	Truck accident	Bedford, Ind.	Unknown	Unknown	Unknown		No
9/13/74	Transformer fell into Duamish River	Seattle, Wash.	283 gal	70-90 gal	Entombed in titan missle	$148,000	Yes
10/25/74	Transformer leak	Elizabeth, N.J.	10-100 lbs	Unknown	Unknown		No
Unknown	Transformer leak	Unknown	10-100 lbs	Unknown	Unknown		No
7/ 5/74	Transformer leak RR	Stanford, Conn.	10-100 lbs	Unknown	Unknown		No
4/16/74	Transformer leak RR	Bet. Phil. & Paoli, Pa.	10-100 lbs	Unknown	Unknown		No
11/18/74	Truck accident	Trion, Ga	130 gals	2,300 drums of contaminated soil	Entombed	$175,000	Yes
12/6/74	Truck accident	Erie, Pa	200 gals PCBs	Unknown	Unknown		No
12/27/74	Truck accident	Lawrence Al	630 gals PCBs	4,500 drums of contaminated soil	Entombed	$275,000	Yes
1/22/75	Truck accident	Rome, Ga	67 gals PCBs	132 drums of soil	Entombed	$5,000	Yes
8/9/75	Leaking drum on dock	Charlestown SC	50 gals PCBs	5 drums of soil	landfill	Unknown	Yes

Figure 10. The 20 identified spills that have involved PCB's.

"OIL AND HAZARDOUS SUBSTANCE LIABILITY"

Sec. 311 of FWPCA, PL92-500, October 18, 1972

* 311(b)(2)(A) Designation of Hazardous Substances

Polychlorinated biphenyls PCB
Aroclor
Polychlorinated diphenyls

* 311(b)(2)(B)(i) Removability Determination

"...cannot actually be removed."

* 311(b)(2)(B)(ii-iv) Rates of Penalty for Non-Removable

PCB's Category A, Unit of Measurement lb - 1,$360/UM

311(b)(3) Non-Harmful Quantities

(being developed)

* 311(b)(4) Harmful Quantities

PCB's Category A, HQ in lb - 1

311(f)(2) Small Facilities Liability Limitations

(being developed)

311(j)(1)(A) Methods of Removal

(being developed)

311(j)(1)(c) Prevention

(being developed)

Figure 11. List of subjects covered in proposed EPA regulation under Section 311 of the Water Act.

the 20 in which PCB's reached the water. This is the Duwamish River incident. The majority of the spills are transportation-related.

EPA is finalizing its proposed regulations under Section 311 of the Water Act, "Oil and Hazardous Substance Liability" (figure 11). At the present time, only four areas will be covered in the proposed regulation and the other four sections will be following next year, we hope.

SOURCES OF POLYCHLORINATED BIPHENYLS IN WISCONSIN

Stanton J. Kleinert*

Abstract

The past and present users of PCB's are discussed and effluent sampling data are presented for municipal wastewater treatment plants and industries in Wisconsin. Data are also presented covering PCB levels in surface waters, fish, sediments, and snow melt. A general assessment of the PCB problem in Wisconsin is made with comments concerning the need for further study and regulation.

My report will summarize the work completed by the Wisconsin Department of Natural Resources and others to determine concentrations of PCB's in fish and to identify discharge sources in Wisconsin. Our interest in PCB's began in the late 1960's, when interfering substances were detected in the gas chromatograms of fish being tested for DDT. Later we were to learn these interfering substances were PCB's.

In 1970, the department collected fish samples along the Mississippi River bordering Wisconsin. Analysis revealed that fish from the Upper Mississippi River between Prescott and Pepin, Wisconsin, commonly exceeded the Food and Drug Administration tolerance level of 5 ppm (ref. 1). During 1971, Lake Michigan fish were collected and later tested in studies conducted by Dr. Veith in a University of Wisconsin study (ref. 2). Mean concentrations of PCB's expressed as Aroclor 1254 (wet weight) in the fish ranged from 2.7 ppm in smelt to 15 ppm in lake trout. Subsequent studies confirmed the presence of PCB's in fish in Lake Michigan and other waters of Wisconsin (ref. 3).

The search for PCB's in water was also underway at this time. Veith and Lee (ref. 4) sampled water from the Milwaukee River in 1969 and sampled effluents from selected municipal and industrial outfalls to the river in 1970. The analysis of water from the Milwaukee River indicated that PCB's were present in the river from West Bend to Lake Michigan and were being discharged through municipal and industrial effluents. In 1971, 11 municipal wastewater treatment plant effluents in Wisconsin were sampled by Dube, Veith, and Lee (ref. 5). Nine of the plant effluents contained PCB's identified as Aroclor 1254 and one contained Aroclor 1248. Studies of the Cedarburg wastewater treatment plant indicated that more than 70 percent of the PCB's coming into the plant were removed during the treatment process and comparatively high concentrations of PCB's were found in the digester and primary settling sludges.

The department surveyed many municipal wastewater treatment plant effluents in Wisconsin from 1972 through 1974. PCB's were detected in concentrations exceeding .05 ppb (the detection level for screening purposes) in more than half of the treatment plant effluents tested, even where there were no suspected industrial sources. In most cases, the discharge of PCB's was well below 1 ppb and .01 pounds per day. However, the higher concentrations of PCB's were found in sewage treatment plant effluents from industrial areas.

Tracing sources of PCB's reaching a large municipal wastewater treatment plant is a difficult and time-consuming task. The department is attempting to trace sources of PCB's reaching treatment systems where the final effluent exceeds 1 ppb. At the present time, we know of only two municipal wastewater treatment plants in Wisconsin which exceed 1 ppb—the Sheboygan and Portage facilities.

The main source of PCB's being discharged to the Portage treatment plant was found to be a facility that had used PCB's in the manufacture of carbonless copy papers prior to the summer of 1971. After ceasing the use of PCB's and after repeated cleanings of the holding tanks of the facility, the company substantially reduced the discharge. Residuals still remain, however, in the sewer system and the sewer sludges, resulting in an effluent of several ppb at the municipal sewage treatment plant. We are continuing to check sources of discharge to the Sheboygan treatment plant.

The department has checked effluents from iron and steel foundries and aluminum foundries. The testing has shown that the cooling water effluents from five to seven aluminum foundries contained PCB's ranging from 11.5 to 335 ppb. Close investigation revealed the common source to be leaking hydraulic fluids containing PCB's, which were used in die cast machines. We are working with company officials to correct the problem. PCB's have been found in the cooling water effluent of only one of nine iron and steel foundries checked to date and these were at a concentration of .9 ppb.

The Department of Natural Resources has tested the effluents of 17 pulp and paper mills for PCB's. The testing has revealed that nine of the mills that recycle wastepapers had measureable discharges ranging from .1 to more than 25 ppb. Mill representatives indicate that the paper industry no longer uses PCB's and those found in wastepapers come primarily from carbonless copy papers that were produced prior to 1972. The old carbonless copy papers were widely used in forms and continue to enter the wastepaper market as old files are discarded. Aroclor 1242 is the principal form found in wastepaper, however, some Aroclor 1254 has also been reported. Because their solubility in water is low, we believe that most of the PCB's discharged from wastepaper mills are absorbed on fibers and other particulate matter. Mill wastewater treatment systems, which effectively remove particulate matter, should also remove PCB's.

---

*Chief, Surveillance, Wisconsin Department of Natural Resources, Madison, Wisconsin.

The electrical industry continues to use PCB's as dielectric fluids in some capacitors and transformers. Although the units are sealed, some fluids may be lost as a result of accidents or disposal practices. In March 1975, the department corresponded with the major electrical companies in Wisconsin to determine current handling practices for capacitor and transformer fluids. The correspondence was followed by visits to many facilities. The companies contacted were aware of the PCB problem, but some were not aware of the recommended Guidelines of the American National Standards Institute (1974) for Handling and Disposal of Capacitor and Transformer Askarels (Fluids) Containing PCB's (ref. 6). We also found that some facilities were storing defective capacitors until a proper disposal method could be found. As a result of this survey, specific guidance was given to Wisconsin electric utilities for the proper handling and disposal of PCB's.

Snow samples were collected early in 1975 to determine if PCB's were deposited on land and water as fallout from the air. Analysis of the snow melt water from Racine, Kenosha, Madison, and Milwaukee revealed concentrations from .17 to .24 ppb. These values suggest that fallout of PCB's from the air may be a principal source of PCB's entering the waters of the State.

PCB's are present in sediments in harbors and streams near industrial areas. The sediments act as a reservoir where PCB's may be released slowly over a long period of time. Sediment samples have tested 3.5 ppm in the Milwaukee River near the Capital Drive Bridge, 9 ppm in Superior Harbor, and 72 ppm in the Fox River below the outfall of the Portage sewage treatment plant.

We have tried to work out a materials balance for PCB's entering the environment using the domestic sales figures provided by the Monsanto Company as well as other data. There are so many pieces missing from the puzzle, though, that our efforts to work out a materials balance have been unsuccessful. Some general comments can be made, however.

1. PCB's have been sold by the Monsanto Company for more than 45 years (ref. 7). The company has reported domestic sales of 795 million pounds of PCB's for the years 1957 through 1974. Aroclor 1242 accounted for 15 percent, Aroclor 1260 accounting for 11 percent, and Aroclor 1248 accounting for 7 percent of the sales. In 1974, the Monsanto Company's domestic sales of PCB's were reported to be 34 million pounds for use in closed electrical systems. In addition, the Office of Toxic Substances, EPA (ref. 8) has reported that foreign sales of PCB's in the United States in 1974 exceeded 375,000.
2. The PCB problem in Wisconsin is a fishery problem caused because residues have accumulated in certain fish in Green Bay and Lake Michigan and the Upper Mississippi River in excess of FDA tolerance level of 5 ppm. Laboratory experiments have shown that fish accumulate PCB's more than 100,000 times the levels present in the water (ref. 9). Therefore, PCB's in the water, even at ppt levels, have significance to the fishery resource.
3. Our data indicate that levels of PCB's in fish in the Upper Mississippi River have declined in recent years. We have not detected a corresponding decline in levels in Lake Michigan fish.

If Lake Michigan water contains an average of 10 ppt PCB's, then there are more than 100,000 pounds of PCB's in solution in the lake waters and there is probably a much larger poundage in the sediments. We have tested the major effluents of both municipalities and industries discharging to the Lake Michigan drainage in Wisconsin and estimate a discharge of about 2 pounds per day or 730 pounds of PCB's per year to Wisconsin's drainage to Lake Michigan. Most of PCB's identified in our testing of major effluents occur in the wastewaters of pulp and paper mills that recycle wastepapers.

Discharges of PCB's from pulp and paper mills that recycle wastepapers will diminish as the mills meet their discharge permit requirements. Wisconsin mills that recycle wastepapers are required to reduce their discharge of suspended solids from 131,000 pounds per day (for calendar year 1973) to 45,000 pounds per day by the 1977 compliance date. Recently one mill in the State, which uses only recycled papers, began operation of a new treatment system that has reduced the discharge of suspended solids from 40,000 pounds per day to 3,000 pounds per day. Tests at this facility revealed 39 ppb PCB entering the treatment system with only 1 ppb being discharged in the final effluent.

4. In our search for sources of PCB's entering the environment, we have not looked closely enough at fallout from the air. Our testing of snow melt suggests that fallout may be contributing much greater amounts of PCB's than are being contributed by industrial and municipal effluents. Trace concentrations in fallout over Lake Michigan and its watershed, which cover 67,900 square

miles, could result in appreciable amounts of PCB's entering the waters of Lake Michigan.

Because PCB's are stable compounds with low vapor pressures, little loss is expected to occur through vaporization from disposal sites where capacitors and other equipment and materials have been disposed and covered with overburden. Entry into the air may be expected to occur at locations where papers are incinerated, at foundries where imported casting waxes containing PCB's are heated to high temperature, and at manufacturing facilities. PCB's adsorbed on fine particulate matter may also be entering the air, as windblown dust.

5. Further information is needed to define the amount of PCB's contributed to Lake Michigan and other waters through past accumulations in sediments. A University of Wisconsin study is currently underway in Southern Lake Michigan; this should provide some of the answers.

The Department of Natural Resources does not have the authority to regulate the sale or use of PCB's, but can adopt effluent standards. The department held hearings on two proposals for effluent limitations for PCB's on August 28-29, 1975. The first proposal would prohibit the discharge of PCB's and the second would limit the discharge to .005 mg/l or 5 ppb. The hearing record is being reviewed by the Natural Resources Board. No final decision has been made on an effluent standard as yet.

On September 10, 1975, Congressman Aspin of Wisconsin introduced a bill (HR 9525) to prohibit the introduction or delivery for introduction into commerce of PCB's. This bill is pending in the House Interstate and Foreign Commerce Committee, Subcommittee on Consumer Protection and Finance. This legislation would become effective 3 years after enactment providing industry with a 3-year changeover period to alternate substances.

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POLYCHLORINATED BIPHENYL USAGE AND SOURCES OF LOSS TO THE ENVIRONMENT IN MICHIGAN

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Abstract

Polychlorinated biphenyls (PCB's) are used in a wide variety of applications including dielectric fluids in transformers and capacitors; plasticizers in paints, inks, plastics, and coatings; hydraulic systems; heat transfer systems; investment casting waxes; and many more. This paper describes industrial uses of PCB's in Michigan and focuses on those uses which were found to be sources of environmental contamination. It describes PCB use figures supplied by several industries and concentrations detected in industrial and municipal effluents and sludges.

INTRODUCTION

Polychlorinated biphenyls (PCB's) are complex mixtures of chlorine-substituted biphenyl compounds and have been produced commercially in the United States since 1929. Their properties of nonflammability, stability, resistance to acids, alkalis, and other caustic chemicals and low volatility under prolonged heating have made them useful in a wide range of industrial products.

Monsanto Chemical Company, the sole U.S. manufacturer of PCB's, recently released production and sales figures through 1974 (ref. 1). These showed that production and sales roughly doubled between 1960 and 1970, with nearly 80 million pounds sold annually in the United States by 1970.

Broadhurst (ref. 2) has summarized many of the uses of PCB's. The five largest uses for PCB's prior to 1970 were dielectric fluids in capacitors, plasticizer applications, transformer fluids, hydraulic fluids and lubricants, and heat transfer fluids. The list of other uses is lengthy.

Because of the widescale usage of PCB's, they have become distributed throughout the world. Since 1967, reports of PCB residues in fish, birds, water, sediments, and other environmental samples have been common. They are of concern in the environment because of their persistence, potential for biological magnification, and chronic toxicity.

By 1971, Monsanto recognized the environmental problems which had developed because of PCB's and initiated a program to phase out sales for all open-ended uses, finally limiting sales to uses in electrical capacitors and transformers. Many officials believed this action would result in rapid reductions in environmental contamination but little evidence of this has yet been documented.

Michigan officials have been studying the PCB situation since 1969 when excessive PCB residues were detected in Great Lakes fish. After development of adequate laboratory capabilities, we instituted a monitoring program for PCB's early in 1971 consisting of a statewide water sampling survey in inland waters and tributaries to the Great Lakes. Supplementary sampling has since been conducted on fish from inland and Great Lakes waters, stream sediments, potable water intakes, sanitary landfill runoff, municipal wastewater treatment plant effluents, and industrial discharges. Limited questionnaire surveys of industrial usage have also been conducted. This paper will report briefly on the industrial uses of PCB's that we have identified in Michigan and focus on those uses which were found to be sources of environmental contamination.

Electrical Applications

Prior to the 1971 Monsanto limitation of sales to the so-called "closed-system" uses, approximately 60 percent of U.S. sales were for electrical capacitor and transformer applications. In 1968, annual capacitor and transformer sales totaled approximately 30 and 16.8 million pounds, respectively (ref. 3).

While losses from the use of PCB's in these applications have been estimated as minimal (ref. 3), incorrect disposal practices and accidental losses from equipment and storage areas do occur and can result in environmental contamination.

In response to an industrial questionnaire sent to a cross section of Michigan industries in 1971, one major power company in Michigan reported an annual use of 562,000 pounds of PCB's (250,000 in precipitator transformers and 312,000 in capacitors). Company records showed annual losses of approximately 300 pounds of PCB liquids to soils from burst capacitors and 12,000 pounds from salvaged capacitors. This fluid from the capacitors was reportedly disposed of through waste haulers. Waste haulers then dispose of these wastes in many ways, including dust control on roads, sale of oils as fuel in low-temperature boilers, and treatment and discharge to municipal sewerage systems. Fluid recovered from precipitator transformers was returned to Monsanto for incineration. In the same industrial questionnaire survey, a smaller power company reported that it had been disposing of approximately 700 pounds of PCB's per year by mixing with other waste oils and using the oils for dust control on their driveway and parking lot.

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In October 1975, a 55-gallon drum of PCB transformer oil on inventory at a Michigan power company developed a leak from a defective seam. Approximately 45 gallons of the fluid soaked into the ground, resulting in more than 100 cubic yards of contaminated soil having to be removed and disposed of in an approved landfill. This situation illustrates the lack of such proper safeguards as diking around storage areas and represents an example of environmental loss from a so-called "closed-system" use.

Most industries other than utility companies using electrical transformers containing PCB's contract either the transformer manufacturer or smaller companies for servicing and do not concern themselves with the problem of disposal of the waste fluids. Frequency of servicing appears to differ between industries, varying anywhere from every 6 months to 10 years.

While water sampling programs in Michigan have failed to identify any electrical applications of PCB's as major point sources of loss to aquatic situations, environmental losses do occur which have the potential of indirectly reaching watercourses through atmospheric fallout or leaching from contaminated soils at spill or disposal sites. Losses directly to water may occur on occasion but would be difficult to detect because of their intermittent nature.

Plasticizer Applications

Plasticizer applications represent the single largest "open-ended" or dissipative use of PCB's. PCB's are or have been used as plasticizers in most countries in a wide variety of consumer products, including paints, inks, copying paper, adhesives, sealants, plastic products, and textile coatings, many of which are traded internationally (ref. 4). Monsanto's U.S. sales figures for 1970 showed a volume of nearly 20 million pounds for plasticizer applications (ref. 1). Sales for this use were discontinued in 1971. Environmental losses from past sales will likely continue for a long period of time.

Papers used in the thermographic, xerographic, or pressure-sensitive copying processes have had PCB's added as plasticizers either in the ink or paper coatings. Recycling of these papers can result in contamination of food packaging materials and other paper products (ref. 3).

Effluents from paper recycling or deinking plants also become contaminated. In Michigan, paper industry effluents have been found to commonly contain from 1 to 10 µg/l of PCB's. Contamination of fish has been identified as high as 110 mg/kg in the Kalamazoo River downstream from Kalamazoo, Michigan, where Michigan's paper industry is primarily centered. Stream sediments in the Kalamazoo area contain as much as 360 mg/kg PCB's; this is believed to be a result of past deinking processes in the area. Lower level contamination continues from processing of recycled paper but most deinking mills have ceased operation over the past 10 years.

While Monsanto stopped sales of PCB's for paper applications in 1971, one Michigan paper company recently reported to the Michigan Department of Natural Resources that several raw products that they purchase still contain PCB's. Two coloring compounds reportedly have 23 and 500 mg/kg PCB's and nearly all of their wood pulp contains from 0.5 to 1.0 mg/kg.

Burning of waste papers containing PCB's, which occurs every day, undoubtedly results in atmospheric losses. PCB's have been detected in snowfall samples in Wisconsin (ref. 5).

The largest quantities of PCB's in plasticizer applications end up in dumps and landfills. Much of the material is in sealed containers or impregnated in plastics and is slowly released to the environment. Nisbet and Sarofim (ref. 6) reported that vaporization directly from paints, coatings, and plastics does occur, with losses as great as 20 percent. Open burning in landfills also releases PCB's to the atmosphere. Another source is loss to leachate from landfills. Samples of surface runoff water from nine landfills in Michigan showed five of the nine samples having PCB concentrations ranging from 0.04 to 0.30 µg/l, while the others contained less than 0.01 µg/l.

In 1970, the U.S. Food and Drug Administration identified milk contamination in Ohio, Georgia, and Florida resulting from use of a PCB-containing sealant in silos (ref. 3). In 1975 the Michigan Department of Agriculture investigated problems from this usage and found 76 dairy herds in Michigan with PCB's in milk ranging from a trace to 14 mg/l on a fat basis. Scrapings from silos on these farms contained PCB's up to 10,000 mg/kg. Eighty additional silos have been identified as having the PCB sealant and have been removed from use until a protective coating can be applied to eliminate transfer of PCB's to the cattle feed.

Hydraulic Fluids

PCB's have found wide usage in hydraulic systems involving extreme pressure and high-temperature conditions. Annual U.S. domestic sales of PCB's for hydraulic uses totaled approximately 8 million pounds in 1970 (ref. 1). In Michigan, the automotive and metal finishing industries have used PCB-containing hydraulic fluids extensively. Industrial inspections and questionnaires documented past usage to have been as much as 878,000 pounds/year in individual plants, with most being collected and disposed of through waste haulers. Large losses of PCB's from these plants to municipal sewers or directly to surface waters have also been identified. Wastewater discharges from identified users of PCB-hydraulic fluids have had concentrations as high as 7.1 mg/l PCB's, indicating losses as great as 30 pounds per day. In a materials balance study, one industry which showed in its records that it was replacing approximately 30,000 pounds PCB's/year in hydraulic systems calculated that 10,000 pounds/year were entering the atmosphere through vaporization losses, 8,000 pounds/year in absorbents placed in landfills, 6,000 pounds/year lost to soils and drainage systems on plant property, 5,000 pounds/year going to waste haulers, with the remainder incinerated.

All industries that we have identified as using PCB's in hydraulic systems have converted to substitute products, mostly phosphate esters. Complete elimination of PCB residues from the systems has proved ineffective even with repeated flushings. Sampling of replacement fluids indicates PCB contamination as high as 100 mg/l. Residues in discharge lines combined with loss of contaminated replacement fluids appear to result in continued low-level discharges. Because of existing inventories and possible purchase from foreign sources or reprocessing companies, it is unlikely that all hydraulic systems not included in our surveys or questionnaires have been switched to replacement fluids. One facility which we investigated had an inventory of 9,000 gallons of PCB fluids in storage.

In summary, Michigan's surveys of hydraulic system usage of PCB's suggests that this has been a major source of environmental contamination and may continue to contribute to the problem to a lesser but still significant degree.

Heat Transfer Systems

PCB's heat transfer fluids have been used in place of steam or superheated water in many industries and large building complexes. They provide advantages of reduced explosion hazards, absence of corrosion problems, and have good heat transfer characteristics (ref. 4). In 1970, Monsanto sold approximately 4 million pounds of PCB's for this application but phased out all sales in 1972 (ref. 3).

From 1971 through 1973, Michigan identified at least 12 facilities discharging PCB's to surface waters or municipal sewers from this usage. Concentrations in some discharges were as high as 5.2 mg/l.

One automotive company in Michigan reported using 34,000 pounds of PCB's per year in heat transfer systems at three of its plants. The waste fluids were reportedly disposed of by industrial waste haulers. Another smaller plant reported loss of 800 pounds in 1972 and that approximately 700 pounds of this total was incinerated, 75 pounds disposed of in dumps, and 25 pounds lost to sewers and drains.

In another Michigan survey, a chemical company complex was found to have an effluent concentration of 35 µg/l resulting from heat transfer system losses. Heat transfer losses are not all from industrial facilities. Sampling of interceptor lines of the Detroit, Michigan, sewerage system resulted in the identification of heat transfer usage from two residential building complexes and one hospital as large sources of PCB input to the system.

Miscellaneous Uses

In 1971, Michigan officials found a PCB concentration of 6.6 µg/l in the waste lagoon of a soap and detergent company. The source was traced back to the company's usage of PCB's as a dedusting agent at 0.7 percent in one of its powdered products. The company had discontinued this usage in June 1970 and the PCB's were apparently leaching from contaminated lagoon sediments.

The investment casting wax industry has been recently identified as another user of PCB's (ref. 7). This casting method, which has largely replaced sand casting for production of low-tolerance-quality metal castings, uses formulated waxes as a basic molding mode. The waxes are injected into metallic dies to form the part shape and then coated with ceramic to form a final mold. The wax is removed by insertion of the mold in an autoclave, which melts the wax. Residues of wax are also secondarily removed by insertion of the molds in a furnace at 1,600° to 1,800° F. During these two procedures, the bulk of the wax is collected and usually recycled but a portion is lost to vaporization, cooling water contact, and poor housekeeping practices.

The waxes in use are obtained from a few major suppliers, one of whom is a major importer of decachlorobiphenyl from an Italian source. We have found twelve firms in Michigan using this casting process and several firms that reprocess their waxes. Our preliminary investigations indicate that most of the waxes contain approximately 20 percent of PCB's or polychlorinated terphenyls (PCT's); one recent sample indicates the range may extend as high as 60 percent content in the wax. The nature of these casting operations, including both operational and storage modes, at many plants indicates strong potential for environmental loss. An effluent sample from one of the Michigan companies using investment casting wax contained 2.5 μg/l of PCB's.

During a telephone survey of 39 industries in Michigan during May 1975, several industries reported use of a toilet hand soap containing 0.05 percent PCB's by weight. Followup on these reports indicated that this product actually contains a chlorinated diphenyl oxide rather than PCB's.

Three industries in the telephone survey reported use of a hydrated lime product that contained about 0.5 mg/kg PCB's. This product was being used for water treatment and in production of glass pellets. No explanation was available on why the lime contained PCB's but it is assumed to be a contaminant rather than an additive.

Concentrations in Municipal Wastes

Because of the diversity of PCB usage, municipal wastewater is likely to receive PCB's from numerous domestic and industrial sources, many of which are listed above. Buckley (ref. 3) estimated that municipal outfalls would likely contribute less than 5 percent (300 tons) of total annual loadings (6,000 tons) to aquatic environments of the North American continent. His estimate was based on an assumed average PCB concentration of 10 μg/l in wastes serving a sewered population of 150 million people producing 130 gallons of sewage per person per day.

Sampling of 58 municipal wastewater treatment plant (WWTP) effluents throughout Michigan in 1973 showed an average concentration of 0.52 μg/l (table 1). This is considerably lower than Buckley's value of 10 μg/l used in his calculations but still exemplifies widespread contamination in municipal wastes, and documents that they are indeed a source of PCB loss to surface waters.

Much of the PCB's entering municipal waste treatment facilities are removed and become incorporated into the waste sludge. Table 2 shows PCB concentrations in the sludges from Michigan municipal plants sampled in 1973. Concentrations are much higher than in the effluents from the same plants. The average for all plants was 15.6 mg/kg, with individual values as high as 350 mg/kg. Disposal of these sludges may add to environmental PCB levels. Sewage sludges are commonly disposed of by incineration, spreading on agricultural land, or placing in landfills.

Discussion

While I have characterized in this report possible and proven sources of environment loss of PCB's in Michigan from several applications, I want to acknowledge that not all industrial effluents tested in Michigan contain measureable PCB levels. We have tested over 900 industrial samples, with approximately 40 percent of effluents sampled containing PCB's above our 0.1 μg/l laboratory sensitivity limit. Twenty-three percent of the industries tested had greater than 0.5 μg/l, 18 percent greater than 1 μg/l, 6 percent greater than 10 μg/l, and 2 percent greater than 100 μg/l. This represents a diversity of industries showing contamination and in many cases the source of effluent contamination could not be determined.

Since new industrial sources of loss are continuously being found, we feel we are far from eliminating the PCB inputs to surface waters by searching for point sources and taking corrective measures on a case-by-case basis. We are far from understanding the impact or comparative contribution of atmospheric losses but we feel these are probably very significant also.

In light of considerable environmental damage and economic impact of PCB contamination, an absence of any apparent decline in environmental concentration with current control methods, the ubiquitous nature of usage, the general lack of adequate disposal facilities, and the indication that atmospheric transport and deposition is also significant, the Michigan Department of Natural Resources urges that a national ban be pursued on all domestic and imported PCB's destined for use other than in transformers and capacitors, and that the critical or essential use of PCB's in transformers and capacitors be immediately and critically reviewed in light of current potential replacement products.

Feeling that existing legislation was inadequate to control PCB's, a group of Michigan legislators introduced a bill on August 14, 1975, to prohibit the manufacture, sale, and use of PCB's in Michigan for all applications other than for transformers and capacitors. The bill would provide for labeling, reporting, and disposal requirements and specifies penalties for violations. The bill calls for a stepwise elimination of PCB's with the sale, manufacture, and use of products containing 25 percent or more PCB's forbidden by January 1, 1976; 10 ppm or more forbidden after January 1, 1977; and 5 ppm or more forbidden after January 1, 1978. Public hearings on the bill are currently being conducted by the Michigan House Committee on Marine Affairs. [The bill was amended and voted out of committee on December 11, 1975. The amendment changed the maximum allowable concentration after January 1, 1977, to 1,000 ppm and 100 after January 1, 1978.]

Table 1. Concentrations of PCB's in the final effluent from wastewater treatment plants throughout Michigan, Spring, 1973. Concentrations in micrograms per liter, dry weight.

City	Date	PCB's 1242	 1254	 1242:1254
Adrian	3/7/73			0.26
Albion	2/22/73		0.34	
Ann Arbor	3/1/73		0.25	
Bay City	3/1/73	3.20		
Battle Creek	2/28/73		<0.10	
Benton Harbor-St. Joseph	2/28/73		0.20	
Brighton	3/1/73			0.33
Cadillac	3/28/73		0.53	
Charlotte	5/1/73		0.34	
Constantine	2/28/73		0.46	
Detroit	2/28/73	2.15		
Dexter	3/1/73		0.23	
East Lansing	5/2/73		0.18	
Escanaba	3/28/73			0.28
Essexville	3/1/73		0.10	
Flint	2/22/73		<0.10	
Gladstone	3/28/73		0.44	
Grand Haven	2/21/73		<0.10	
Grand Rapids	2/21/73		1.05	
Holland	2/21/73		<0.10	
Houghton-Hancock	3/27/73		0.18	
Iron Mountain-Kingsford	3/28/73		0.69	
Ironwood	3/27/73			0.29
Jackson	2/22/73		<0.1	
Kalamazoo	2/29/73			1.12
L'Anse	3/27/73			0.31
Lansing	4/18/73		0.22	
Manistique	3/28/73		0.63	
Marquette	3/27/73		0.29	
Marshall	2/22/73		0.15	
Menominee	2/28/73		0.57	
Midland	3/1/73		<0.10	
Milford	3/8/73			2.20
Monroe	5/4/73		<0.10	
Mt. Clemens	3/8/73			2.90
Mt. Pleasant	3/28/73		0.29	
Muskegon	2/28/73		0.88	
Muskegon Heights	2/28/73		0.18	
Niles	2/20/73		0.48	
Norway	3/28/73		<0.10	
Owosso	2/21/73		0.12	
Pontiac (Auburn)	3/12/73			0.60
Pontiac (E. Blvd.)	3/12/73			0.54
Port Huron	3/8/73			1.80
Saginaw	3/1/73			0.77
Sault Ste. Marie	3/28/73		0.73	
St. Ignace	3/28/73		0.31	
Three Rivers	2/21/73		<0.10	
Traverse City	3/7/73		<0.10	
Trenton	2/28/73		<0.10	
Warren	2/27/73		<0.10	
Wayne County	2/28/73		0.40	
Wyoming	2/21/73		0.22	
Ypsilanti	2/28/73			0.31
Ypsilanti Twp #1	3/1/73		<0.10	
Ypsilanti Twp #2	3/1/73		<0.10	

[a] Concentrations based on best fit of three standards: Aroclor 1242, Aroclor 1254, or a mixture of Aroclor 1242 and 1254 in a 1:1 ratio.

Table 2. Concentrations of PCB's in the sludge from fifty-seven municipal wastewater treatment plants in Michigan, Spring 1973. Concentrations in milligrams per kilogram, dry weight.

City	Date	PCB's		
		1242	1254	1242:1254
Adrian	3/7/73			24.0
Albion	2/22/73		1.5	
Ann Arbor	3/1/73		1.1	
Bay City	3/1/73	352.0		
Battle Creek	2/28/73		2.8	
Benton Harbor-St. Joseph	2/28/73		13.8	
Brighton	3/1/73		<0.1	
Cadillac	3/28/73		<0.1	
Charlotte	5/1/73		6.8	
Constantine	2/28/73		2.1	
Detroit	2/28/73	32.1		
Dexter	3/1/73		3.2	
E. Lansing	5/2/73		4.6	
Escanaba	3/28/73		5.9	
Essexville	3/1/73		3.9	
Flint	2/22/73		6.3	
Gladstone	3/28/73		4.1	
Grand Haven	2/21/73		4.1	
Grand Rapids	2/21/73		11.8	
Holland	2/21/73		0.8	
Howell	4/26/73		15.0	
Houghton-Hancock	3/27/73		5.5	
Iron Mountain-Kingsford	3/28/73		9.5	
Ironwood	3/27/73		5.2	
Jackson	2/22/73		3.0	
Kalamazoo	2/29/73	23.3		
L'Anse	3/27/73		4.4	
Lansing	4/18/73		5.3	
Manistique	3/28/73		1.5	
Marquette	3/27/73		2.8	
Marshall	2/22/73		3.9	
Menominee	2/28/73		4.2	
Midland	3/1/73		2.9	
Milford	3/8/73			50.5
Monroe	5/4/73		3.3	
Mt. Clemens	3/8/73	175.0		
Mt. Pleasant	3/28/73		6.5	
Muskegon	2/28/73		12.7	
Muskegon Heights	2/28/73		11.0	
Niles	2/20/73		7.8	
Norway	3/28/73		<0.1	
Owosso	2/21/73		2.0	
Pontiac (Auburn)	3/12/73			12.1
Pontiac (E. Blvd.)	3/12/73			9.2
Port Huron	3/8/73			9.2
Saginaw	3/1/73		5.0	
Sault Ste. Marie	3/28/73		2.4	
St. Ignace	3/28/73		1.5	
Three Rivers	2/21/73		4.1	
Traverse City	3/7/73		1.8	
Trenton	3/28/73		<0.1	
Warren	2/27/73		<0.1	
Wayne County	2/28/73	2.0		
Wyoming	2/21/73		0.58	
Ypsilanti	2/28/73		2.0	
Ypsilanti Twp #1	3/1/73		<0.1	
Ypsilanti Twp #2	3/1/73		<0.1	

[a] Concentration based on best fit of three standards: Aroclor 1242, Aroclor 1254, or a mixture of Aroclor 1242 and 1254 in a 1:1 ratio.

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GENERAL DISCUSSION OF SESSION II

CHAIRMAN GARRETT: I applaud all the speakers for their interesting insights in these matters. At this point I'd like to call for questions from the floor.

MR. DON SKINNER (Environment Canada, Ottawa, Canada): I am from Canada, and I would like to address myself to Mr. Tom Kopp. Tests going on at St. Lawrence Cement in Ontario, to set the record straight, are being financed by the Government of Canada and the Government of Ontario. The participation in that program other than the suppliers of the waste is still up to EPA?

MR. THOMAS E. KOPP (Environmental Protection Agency, Washington, D.C.): Yes, sir.

MR. HOWARD A. FORMAN (United Electrical Workers, New York, New York): I would like to address my question to Mr. Kopp. You had mentioned how EPA is surveying the use and disposal of PCB's. I know that at least our use was not surveyed, we represent a very valuable source of information on PCB use, especially regarding capacitor plants and transformer plants. Does EPA plan to survey and try to get information from the various labor unions involved in the electrical manufacturing industry?

MR. KOPP: That is a very interesting idea, to survey the unions. I will discuss this with our Office of Enforcement. We'll get together and discuss it and if you'll let me have your name and address, we'll get back to you.

CHAIRMAN GARRETT: I have one question I'd like to ask Bob Durfee. There may be some question that may arise from this, but Bob, would you please describe the differences between the two terms mentioned just now, Aroclors and askarels.

DR. ROBERT L. DURFEE (Versar, Inc., Springfield, Virginia): Well, Aroclors are a trademark of Monsanto Company, and askarels are a class of fluids which by their nature do not form combustible products during an electrical breakdown in a dielectric system. There are several types of askarels; among them is the metalated PCB.

MR. RICHARD FERRY (Bio International, Inc., Woods Hole, Massachusetts): You told us about filtration of PCB's. Do you have a handle on the quantity of PCB's and how they are disposed of?

DR. DURFEE: The disposal of all solid wastes containing PCB's is by landfill. There is no incinerator operating at the present time that will accomodate solids and destroy the PCB. As far as the quantities are concerned, I really do not know what the total is going to be. If you say that all transformer fluids are cleaned up once every 5 years or something like that, then perhaps the quantity will be less than 1 percent, and this will be replaced by fresh PCB's.

MR. WILLIAM H. BUSCH (Illinois EPA, Springfield, Illinois): This is not so much a question as a concern. It seems like there is another open-ended source of PCB's in that most electrical capacitors, the small ones, end up as a throw-away item in home entertainment products.

Television sets, radios, and motor-starting capacitors ultimately fail and are discarded in landfills. It seems like we should be giving more attention to groundwater around landfills. This was touched on earlier today.

CHAIRMAN GARRETT: We have been considering this aspect.

MR. JACK MEDVILLE: Dr. Durfee described the control of U.S. manufacture of PCB's. Does the presentation indicate that the United States has shut off foreign manufacturers of PCB's?

MR. KOPP: We have no legal authority to control importation of PCB's into the United States.

MR. MEDVILLE: I will phrase the question differently. We have received here a number of figures of the amount that is going out and being reentered each year from U.S. manufacturing; is there an outlet on what is coming in from foreign nations?

MR. KOPP: The information we get is from the Bureau of Customs. That is maintained confidential, which is upon their request. Only bulk PCB's are reported. The amount of PCB's entering in equipment, such as transformers, we have no idea, but we are trying to identify the importers of capacitors and transformers to the United States.

MR. MARK WILSON: I am from Canada. I am not addressing my question to any particular member. Does anyone know if PCB's are still used in aircraft, and secondly, what will the offshore oil lines do? Are they bringing their own European hydraulic facilities in?

MR. KOPP: We have thought about this a great deal. We have also thought about ships that are coming into the United States from overseas, because of their hydraulic systems. We do not know of any PCB's being used in aircraft except in capacitors.

These things we are trying to get information on, and we hope to get more consultation with the members of the Organization of Economic Cooperation Development.

20 November 1975

Session III:

ENVIRONMENTAL FATE AND OCCURRENCE

Ian C.T. Nisbet, Ph.D.*
Session Chairman

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INTRODUCTORY REMARKS

Ian C. T. Nisbet, Ph.D.

This is Session III on Environmental Fate and Occurrence of PCB's. We have eight papers on residue levels of PCB's in the environment, followed by four papers on transport and transformation in the environment.

We are very short of time to present 12 papers in 4 hours, so I am going to be pressing on the speakers to cut down the time they spend as much as possible. I hope you will forgive them if they omit supporting details of some of what they say. We will not be able to entertain any questions or discussion until the end of the session.

I have been asked to introduce this session by explaining what we knew about PCB's in 1971 when Monsanto first made its voluntary restrictions on sales, so that we can see what has been learned in the last 4 years. At the end of 1971, Adel Sarofim and I reviewed the data that were available on the occurrence of PCB's in the environment and tried to construct a transport model.

We wrote two papers; a preliminary version was published in *Environmental Health Perspectives* (1:21-38, 1972), and a considerably fuller version came out a few months later in the journal *Environmental Research* (5:249-362, 1972), incorporating additional data, corrections, and a comparison of our transport model with observations of residue levels in the environment. It seems that almost no one has read this second paper in *Environmental Research*, the main reason probably being that it cost $8. The moral of the story for us scientists is that if we want our work to be read and quoted, we have to release large quantities of reprints into the environment, and direct them down environmental gradients into compartments where they will have maximum biological impact.

In these papers we did four things. The first was to review the data on production of PCB's in the United States and their uses. We reviewed what data were available on releases of PCB's into the environment, both deliberate releases and accidental ones. We filled in this information with what was essentially guesswork about the remaining releases into the environment, based on our knowledge of the lifetimes of products containing PCB's, disposal practices, and the likely behavior of PCB's in the environment, including their behavior during incineration. Our conclusion was there were three major routes of release of PCB's into the environment as of 1971. The most important, amounting to about three quarters of the total, we believed would be deliberate discarding of PCB's and products containing them, mostly into dumps and landfills. Much of this discarded material would be incorporated in sealed containers and plastic. We had no knowledge whatsoever whether the material put into dumps was leaching out or not.

The second most important route, accounting for two-thirds to three-quarters of the remaining disposal, we believed was into water. This was primarily attributable to losses of industrial fluids, including hydraulic fluids, dielectrics, heat exchange fluids, fluids in compressors, and disposal of scrap fluids from manufacturing operations.

The third route of release, which we thought accounted for the balance of the total, was into the air. We thought that there were several substantial contributions to release to the atmosphere, the major one being evaporation from plasticized products. We thought there were probably also substantial releases from incomplete burning in dumps and poorly operating incinerators, together with some releases from burning of scrap products. There was also known to be some release into the environment from dedusting agents containing PCB's used on roads and parking lots.

The second thing we did was to review what was known about transport of PCB's in the environment. This appeared to fall into two major categories, transport in air and transport in water. We guessed that most aerial transport was due to passive movement of PCB's trapped on airborne particulates, eventually returning to earth in rain and dustfall. We reviewed transport in water with particular respect to transport downstream into the Great Lakes and the sea. We thought there were a number of different methods of transport, all of which were significant. These included transport in solution, movement of sediment containing PCB's, dredging of contaminated sediment followed by dumping at sea, deliberate dumping at sea of industrial waste, dumping at sea of sewage sludge, transport in sewage to ocean outfalls, leaks directly into estuaries from coastal industries, and finally disposal from ships.

The third thing that we did was to compare our estimates of releases and transport to levels that had been reported in the environment. We found five major discrepancies.

1. Surveying the environment, we could not account for more than a very small fraction of PCB's that were being released or had been released in the past.

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We concluded that most of the PCB's that had been discarded in the past were still in dumps and landfills.

2. The amount that we estimated as being released into freshwater was very much larger than anything we could estimate as being transported to the sea. We concluded that PCB's were accumulating in sediments in rivers and lakes. We also thought that they were accumulating in sediments at sea on the continental shelf.
3. Most environmental residues were of pentachlorobiphenyls and higher chlorinated species, although about half of the releases into the environment at that time and in the past had been of tetrachlorobiphenyls and lower chlorinated species. Our conclusion was that the tetrochlorobiphenyls and lower chlorinated species were being rapidly degraded in the environment. This conclusion was based on the fact that we could not find any evidence for differential transport and dispersion of the lower isomers away from points of release.
4. PCB levels in the ocean and in marine organisms—plankton, fish, and birds—were much higher than could be accounted for by aerial transport. We suggested speculatively that the major source of PCB's in the ocean might be marine hydraulic fluids.
5. There were higher residues in terrestrial birds than could be accounted for by considering known dispersive uses.

Accordingly we posed in 1972 six major questions about PCB's in the environment:

1. What happens to chlorinated dibenzofurans in the environment? Are they released, are they persistent, and are they bioaccumulative?
2. Are the PCB's being discarded into dumps going to stay there or are they going to leak out in the future? This is perhaps one of the biggest environmental questions to be solved, because our estimate was that over the past few decades something of the order of 300,000 tons of PCB's had gone into dumps and landfills.
3. Are the PCB's that are now in sediments in freshwaters and the shallow sea going to be covered up and sequestered away from us, or are they going to be continually recycled back into the environment in future years?
4. Is it correct that the tetrachlorobiphenyls and the lower chlorinated species really are rapidly degraded, and is there significant human exposure to these lower chlorinated species?
5. What is the extent of human exposure, both the average and the extremes? We suggested some numbers in our papers, but we concluded that they were very ill-defined and that the extent of human exposure was very variable.
6. Finally, what would be the effect of Monsanto's curtailment of dispersive uses in 1971? We conjectured that this action would cut out only part of the influx into the environment, and that it would be a long time before some compartments in the environment show decreases in environmental levels.

These are the questions which I think will be addressed in part by our 12 speakers this morning. In my summary statement, I will come back and see how far we have come in answering those questions in the last 4 years.

RESIDUES OF POLYCHLORINATED BIPHENYLS IN THE GENERAL POPULATION OF THE UNITED STATES

Frederick W. Kutz, Ph.D., and S. C. Strassman*

Abstract

Residues of polychlorinated biphenyls have been found in human adipose tissue and in milk collected from the general population of the United States. In a national survey of human adipose tissue during fiscal years 1973 and 1974, 35.1 and 40.3 percent, respectively, of the tissue collected contained levels of 1 ppm or more of polychlorinated biphenyls on a wet-weight basis. Electron capture-gas chromatographic analysis of this tissue revealed that the compounds found in adipose tissue were most comparable to those prevalent in Aroclor 1254 and Aroclor 1260. Additionally, semiquantitative estimation of these residues was accomplished by thin-layer chromatography. Evidence from gas-liquid chromatography-mass spectrometry indicated that the most frequently encountered polychlorinated biphenyl residues were penta-, hexa-, and heptachlorobiphenyl compounds.

Human milk samples collected in selected counties of Arkansas and Mississippi were found to contain trace quantities (<1 ppm) of polychlorinated biphenyls on a wet-weight basis.

Human exposure to polychlorinated biphenyls may come from direct contact with industrial products containing these compounds, from association with contaminated environmental components or from many combinations of the two. Intake of these compounds may occur by three routes: (1) ingestion, (2) respiration, and (3) absorption through the skin and mucous membranes. Most probably, all contribute to the total body burden of these chemicals; none of these routes of exposure can now be identified as the most important.

Polychlorinated biphenyls have been found in human adipose tissue collected for the National Human Monitoring Program as reported by Yobs (ref. 1). This program is a continuing ambient monitoring activity which functions to determine, on a national scale, the exposure of the general population to pesticides and to assess changes in these parameters when they occur. Polychlorinated biphenyls, in addition to certain organochlorine pesticides, are detected in human adipose tissue following a multiresidue analytical procedure stipulated by the National Human Monitoring Program for Pesticides.

The objective of this paper is to report the findings of polychlorinated biphenyls during the human monitoring surveys conducted in fiscal years 1973 and 1974. These results are compared to those obtained in the 1971 survey. Additionally, the results of the analysis of human milk for polychlorinated biphenyls are presented. Data from gas-liquid chromatography-mass spectrometric analysis of the polychlorinated biphenyls in human adipose tissue and milk are also reported.

Early confirmation of polychlorinated biphenyls in human depot fat was accomplished in 1967 by Widmark (ref. 2) using combined gas chromatography mass spectrometry. An article by Biros et al. (ref. 3) reported that two human adipose tissues examined by combined gas chromatography mass spectrometry were shown to contain substantial quantities of polychlorinated biphenyls ranging from pentachlorobiphenyl to decachlorobiphenyl and included at least 14 isomers and chlorine homologs. These samples, supplied by the National Human Monitoring Program for Pesticides, were selected because of indications of unusually high levels of these chemicals.

One of the laboratories routinely engaged in human tissue analysis, under contract to the National Human Monitoring Program for Pesticides, published data regarding the polychlorinated biphenyl content of human adipose tissue (ref. 4). According to this study, 41 to 45 percent of the general population had levels of 1 ppm or more of polychlorinated biphenyls with isomers contained in Aroclors 1254, 1260, 1262, and 1268. The presence of these compounds, ranging from pentachlorobiphenyl to decachlorobiphenyl, was confirmed in three samples by combined gas chromatography mass spectrometry.

Polychlorinated biphenyls were found in measurable amounts in 31.1 percent of 637 samples of human adipose tissue collected from the general population as part of the Human Monitoring Survey during 1971 (ref. 1). Sample collection involved 18 states and the District of Columbia. Positive samples were obtained from each political jurisdiction sampled.

Residues of these compounds have also been detected in other human tissues and milk. In a study of human milk collected in Colorado, 8 of 40 samples contained residues of polychlorinated biphenyls ranging from 40 to 100 ppb (ref. 5).

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METHODS AND MATERIALS

Samples of adipose tissue from individuals of the general population were obtained through the cooperation of pathologists and medical examiners in 75 selected collecting sites throughout the contiguous 48 States.

Sampling: A stratified random design, with sampling proportioned to populations, was followed for selecting cities in which tissue samples were collected. The strata were the nine census divisions as designated in the 1970 Census; the sampling units in each census region were cities with populations in excess of 25,000 people. The number of cities designated within each census division was based on population. The cities which served as collecting sites are shown in figure 1.

This design provided samples which are statistically representative of the general population. For each collection site, an annual sample quota was established to reflect the demographic distribution in that census division. The sample quota in each of these groups was allocated proportionally according to the age, sex, and race distribution, as determined by the 1970 Census.

Adipose tissue was collected by cooperating pathologists and medical examiners from postmortem examinations and from specimens which had been removed during therapeutic surgery. Thus, tissues were received from patients having pathological conditions as well as those dying from sudden acute illness or from trauma. Information recorded for each tissue sample included age, sex, race, and pathological diagnosis. Geographic residence was assumed to be in the general location of the hospital. Since the objective of the program was to reflect the pesticide and other pollutant burden in the general population, samples were not collected from cases in which a diagnosis of pesticide poisoning was suspected or known, from cachectic patients, or from patients who had been institutionalized for extended periods. Further details of the program were presented by Yobs (ref. 6).

Chemical Analysis: Samples were analyzed chemically by contract laboratories using only methodologies specified by the program. All laboratories were required to maintain acceptable performance levels in an interlaboratory quality assurance program established and moderated by the EPA Pesticides and Toxic Substances Effects Laboratory, Research Triangle Park, N.C. 27711. This laboratory also provided technical assistance for the analytical portion of the program.

Samples were analyzed for selected chlorinated hydrocarbon insecticides (table 1), and polychlorinated

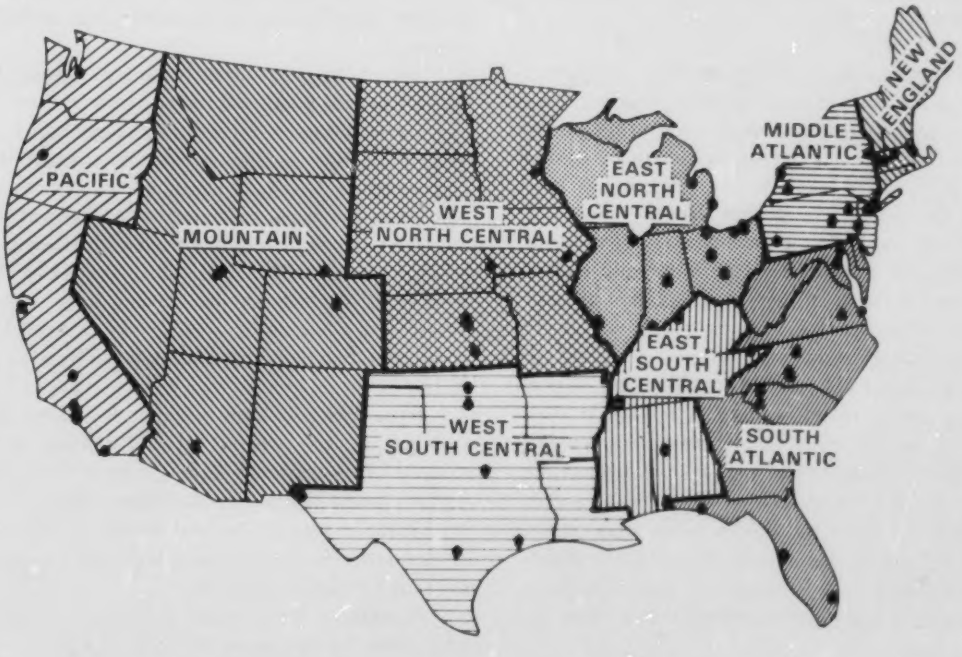


Figure 1. Map of the United States showing census divisions and collection sites for the National Human Monitoring Program for Pesticides.

Table 1. List of chemicals detectable in human adipose tissue

- o,p'-DDT
- p,p'-DDT
- o,p'-DDE
- p,p'-DDE
- o,p'-DDD
- p,p'-DDD
- α-BHC
- β-BHC
- lindane
- δ-BHC
- aldrin
- dieldrin
- heptachlor
- heptachlor epoxide
- endrin
- mirex
- oxychlordane
- trans-nonachlor
- polychlorinated biphenyls
- hexachlorobenzene

biphenyls, using a modified Mills-Olney-Gaither procedure (ref. 6). Gas-liquid chromatography with electron capture detection was employed for basic compound identification and quantification. The analytical determinative procedure for polychlorinated biphenyls in tissue involved: (a) extraction, partitioning, and Florisil column chromatographic purification of the residues together with the chlorinated hydrocarbon pesticides present in the tissue sample, and (b), thin layer chromatographic (TLC) semiquantitative estimation of residues in the purified extract following elimination of interferences from *o,p'*- and *p,p'*-DDE, DDD, and DDT residues by chemical conversion to the respective isomer of dichlorobenzophenone. The details of the TLC procedure included treatment of the extract with ethanolic potassium hydroxide to effect dehydrochlorination of DDT and its analogs to DDE. This was followed by oxidation with chromium trioxide in acetic acid to convert DDE residues to dichlorobenzophenone, which may be conveniently separated from the polychlorinated biphenyl residues by the TLC procedure. The plates were coated with silver nitrate-impregnated aluminum oxide G and developed with 5 percent benzene in hexane. Ultraviolet visualization of the eluted polychlorinated biphenyl residues and semiquantitative comparison of sample spot intensity with those observed for standard materials completed the polychlorinated biphenyl determination.

A total of 140 human adipose tissue sample extracts from the general population have been subjected to analysis by combined gas chromatography-mass spectrometry (GC-MS) to confirm and identify polychlorinated biphenyl components stored in human tissue. Levels of total polychlorinated biphenyls in the samples based on TLC determination ranged from 0.4 to 3.5 ppm. The adipose tissue samples had been extracted and subjected to the purification procedures described earlier. Conversion of the DDT-type residues to dichlorobenzophenone was effected by the combined dehydrochlorination, oxidation, chemical procedures. A final Florisil column chromatographic step was included to further purify the polychlorinated biphenyl residues and remove the dichlorobenzophenones present at relatively high levels in the extract.

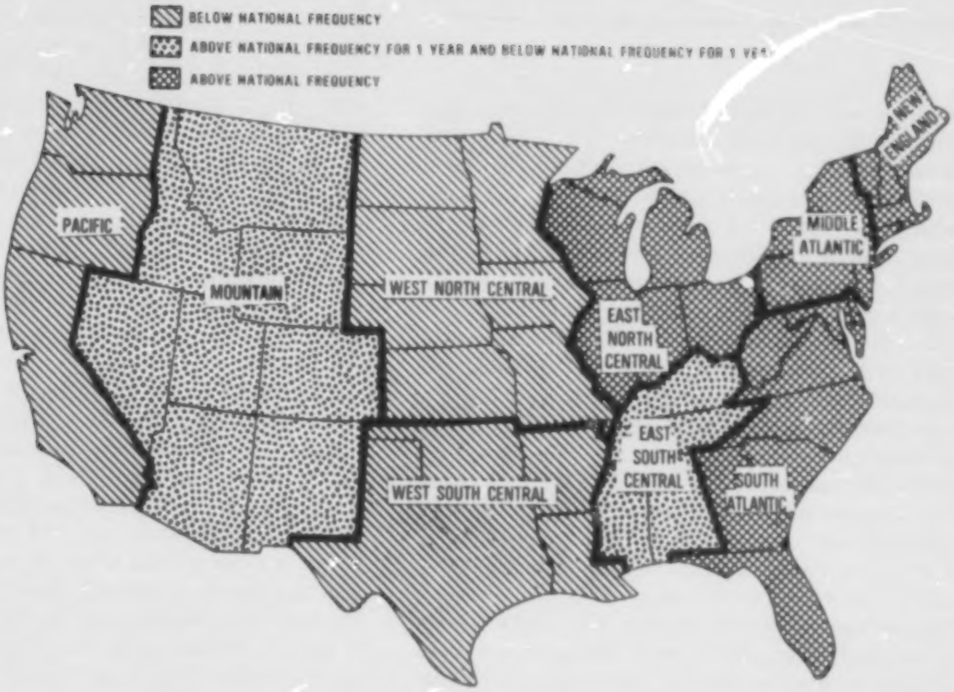
RESULTS AND DISCUSSION

The levels of polychlorinated biphenyls found in general population samples of adipose tissue during fiscal years 1973 and 1974 are presented in table 2. Results presented by Yobs (ref. 1) are included for comparative purposes. During fiscal years 1973 and 1974, 35.1 and 40.3 percent, respectively, of the tissues collected contained levels of 1 ppm or more of polychlorinated biphenyls on a wet-weight basis. These frequencies are slightly higher than the 32.5 percent figure reported by Yobs (ref. 1) and are slightly lower than those (41-45 percent) reported by Price and Welch (ref. 4). The percentage of tissues which contained quantifiable amounts of polychlorinated biphenyls appears to be remaining relatively constant. However, there does seem to be an increase in the percentage of tissues which contained trace levels of this chemical although the limit of detection (1 ppm) remained constant. It should be interesting to determine whether these trace values will be converted to quantifiable amounts as the exposure of the general population continues over time.

National frequencies for fiscal years 1973 and 1974 were compared to frequencies for each census division and are presented in figure 2. The New England, Middle Atlantic, South Atlantic, and East North Central Census Divisions had frequencies of quantifiable levels exceeding the national frequencies for both survey years. The Mountain and East South Central Census Divisions had levels higher than the national levels for one survey year and lower for the other survey year. The Pacific, West North Central, and West South Central had levels lower than the national levels for both survey years.

Table 2. Levels of polychlorinated biphenyls in human adipose tissue

Data source	Sample size	Percent nondetected	Percent < 1 ppm	Percent 1-2 ppm	Percent > 2 ppm
Yobs, 1972	688	34.2	33.3	27.3	5.2
FY 1973 Survey	1277	24.5	40.2	29.6	5.5
FY 1974 Survey	1047	9.1	50.6	35.4	4.9



▨ BELOW NATIONAL FREQUENCY
▨ ABOVE NATIONAL FREQUENCY FOR 1 YEAR AND BELOW NATIONAL FREQUENCY FOR 1 YE
▨ ABOVE NATIONAL FREQUENCY
PACIFIC
MOUNTAIN
WEST NORTH CENTRAL
WEST SOUTH CENTRAL
EAST NORTH CENTRAL
EAST SOUTH CENTRAL
SOUTH ATLANTIC
MIDDLE ATLANTIC
NEW ENGLAND

Figure 2. Map of the United States depicting census divisional frequencies of polychlorinated biphenyls.

The most frequently encountered polychlorinated biphenyl residues in the human adipose tissue extracts examined by combined gas chromatography-mass spectrometry were the penta-, hexa- and heptachlorobiphenyl compounds.

As polychlorinated biphenyls enter a biological system, the less chlorinated isomers are apparently either metabolized or excreted. These biochemical reactions alter the original Aroclors, so that electron capture chromatograms of these compounds from biological systems do not exactly match available laboratory reference standards. However, it can be noted that results do indicate that the polychlorinated biphenyls found in human adipose tissue most closely resemble Aroclor 1254 and Aroclor 1260.

The National Human Monitoring Program also collected and analyzed 57 samples of human milk from selected areas of Arkansas and Mississippi. The survey design was related to rice culture areas in which the herbicide 2,4,5-T was used. All of these samples contained trace amounts of polychlorinated biphenyls, the presence of which were confirmed by combined gas chromatography-mass spectrometry in a composite sample.

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PCB RESIDUES IN HUMAN ADIPOSE TISSUE AND MILK

Donald L. Grant, Ph.D.*, J. Mes*, and R. Frank**

Abstract

Surveys of PCB residues in human adipose tissue suggest that females have lower residues than males, that residents of Central Canada (Manitoba and Saskatchewan) have lower levels than the rest of Canada, and that the majority of Canadians have an adipose tissue residue of 1 to 2 mg/kg. PCB residue in human milk of Ontario residents was found to be approximately 1 mg/kg fat.

Data on PCB residues in adipose tissue obtained in a national survey by Health and Welfare Canada and data obtained in a survey of residents of Ontario will be presented. Human milk PCB residue data obtained in Ontario will also be presented. The Ontario surveys were carried out by the Ontario Ministries of Health and of Agriculture and Food.

The adipose tissue samples were collected at autopsy. In all surveys, organochlorine pesticides as well as PCB residues were determined. The PCB and organochlorine pesticide residues were separated by column chromatography prior to gas-liquid chromatography and quantitation with an electron capture detector.

PCB residue data for human adipose tissues obtained in the Health and Welfare Canada survey are presented in tables 1 and 2.

All adipose tissues had detectable levels of PCB's and 30 percent of the samples had PCB residues greater than 1 mg/kg. The range of residues present was 0.11 to 6.60 mg/kg. There are some regional differences; 49 percent of the adipose tissues samples collected in Ontario had PCB residues greater than 1 mg/kg, while only 9 percent of the samples collected in Manitoba and Saskatchewan (Central Region) had PCB residues greater than 1 mg/kg. The other three regions were very similar with approximately 25 percent of the samples having residues greater than 1 mg/kg.

The mean PCB residue for all adipose tissues collected was 0.907 mg/kg (table 2). The residue present in adipose tissue from males was greater than that present in adipose tissue from females. Residues present were highest in samples collected in Ontario, with means of 1.165 and 0.859 mg/kg for males and females, respectively, and lowest in samples collected in Central Canada (Manitoba and Saskatchewan), with means of 0.621 and 0.377 mg/kg for males and females, respectively. Although differences were noted when the data were broken down (0 to 25 years, 26 to 50 years, 50+ years) to study the effect of age, these differences were not statistically significant.

The results of the surveys carried out by the Ontario government during the years 1969-1974 (table 3) suggest that the mean PCB residue in human adipose tissues collected in Ontario is higher than that reported in the survey by Health and Welfare Canada. However, the Ontario survey results are expressed on a fat basis, while the Health and Welfare Canada surveys are presented on a whole wet tissue basis. The adipose tissue contained approximately 80 percent extractable fat. Although the 1969-70 mean residue value is half that reported for the years 1971-72 and 1973-74, the authors believe this is due more to method refinement than to an actual doubling of PCB residue.

The PCB residue data obtained for the years 1971-72 and 1973-74 are broken down so that an age comparison for PCB residues can be made (table 4). Although the results have not been statistically analyzed, the 21- to 40-year age group did show somewhat lower residues than the 41- to 60- and 61- to 80-year groups.

Residue surveys were also carried out during the period (1969-1974) on human milk. The residue results suggest no change in PCB residue concentration during the years 1969-74 (table 5).

In summary, surveys of PCB residues in human adipose tissue suggest that females have lower residues than males, that residents of Central Canada (Manitoba and Saskatchewan) have lower levels than the rest of Canada and that the majority of Canadians have an adipose tissue residue of 1 to 2 mg/kg. PCB residue in human milk of Ontario residents was found to be approximately 1 mg/kg fat.

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Table 1. PCB residues in human adipose tissue

Region	Samples with PCB ≥ 1 mg/kg	Total No. samples	Percent samples with PCB ≥ 1 mg/kg
Ontario	28	57	49
Quebec	11	50	22
Atlantic	4	16	25
Central	2	22	9
Western	6	27	22
Canada	51	172	30

Table 2. PCB residues (mg/kg) in human adipose tissue

Sex	Atlantic	Quebec	Ontario	Central[a]	Western[b]	Canada
M	0.758(13)	1.125(30)	1.165(38)	0.621(11)	0.977(19)	1.020(111)
F	0.593(3)	0.723(19)	0.859(17)	0.377(11)	0.684(7)	0.685(57)
M&F	0.727(16)	0.969(49)	1.070(55)	0.499(22)	0.898(26)	0.907(168)

[a] Central = Saskatchewan and Manitoba.

[b] Western = Alberta and B. C.

Table 3. PCB residues (mg/kg)[a] in human[b] adipose tissue

Year	Samples	Range	Mean
69-70	20	1.0-2.0	1.2
71-72	282	ND-18	2.5
73-74	129	0.6-11.0	2.3

[a] Fat basis.

[b] Ontario residents, Holdrient, et al. (ref. 1).

Table 4. PCB residues (mg/kg)[a] in human[b] adipose tissue

	Age		
Year	21-40	41-60	61-80
1971-72	0.5-6.0	ND-8.0	0.5-10.0
Mean	2.2	2.6	2.6
Samples	40	96	119
1973-74	0.6-6.0	0.7-7.0	0.6-11
Mean	2.1	2.2	2.5
Samples	13	47	54

[a] Fat basis.

[b] Ontario residents, Holdrient et al.

Table 5. PCB residues (mg/kg)[a] in human[b] milk

Year	Samples	Range	Mean
1969-70	43	0.7-1.2	1.0
1971-72	34	0.2-3.0	1.2
1973-74	19	0.1-2.5	1.2

[a] Fat basis.

[b] Ontario residents, Holdrient et al.

LEVELS OF PCB's IN THE U.S. FOOD SUPPLY

Charles F. Jelinek, Ph.D., and P. E. Corneliussen*

Abstract

The Food and Drug Administration has taken regulatory action since late 1969 to protect the consumer from foods found to contain hazardous levels of PCB's. It has monitored for PCB's in (1) specific products such as milk, (2) raw agricultural products, (3) commercial fish at the wholesale levels, and (4) the total diet. The foods examined were commercial products, essentially all in interstate commerce. Data will be presented on the concentration of PCB's obtained in different food commodities from 1969 through mid-1975. These data show that there has been a significant decrease in PCB levels in all foods with the exception of fish, where no particular trend has been noted. In the case of fish, almost all lots containing more than the FDA tolerance level of 5 ppm in the edible portion originated from the Great Lakes area. It is concluded that the procedures instituted to exclude the use of PCB's in the "open" applications have been effective, but that more needs to be done to prevent their entry into the aquatic environment.

The Food and Drug Administration (FDA) has taken regulatory actions since late 1969 against foods or feeds containing hazardous amounts of PCB's. The purpose of this paper is to summarize the data FDA has obtained on PCB levels in the different types of foods, and to cite the trends which have taken place since our first actions.

Table 1 summarizes the highlights from FDA's analysis of approximately 15,000 samples from November, 1969, through June, 1971. It will be noted that fish, cheese, milk, eggs, and byproducts used in animal feeds were the main commodities contaminated by PCB's. Such foods of animal origin were frequently found to contain significantly high levels of PCB's.

This table does not include data developed by the United States Department of Agriculture (USDA) in its surveillance of meat and poultry during this period. In addition, the data do not reflect the food contamination that occurred later as a result of the leakage of PCB-containing heat exchange fluid into a pasteurized fish meal, which in turn was fed to poultry and catfish. This was by far the most serious incident of PCB contamination of our food supply and led to very extensive regulatory actions by FDA and USDA, such as seizures and recalls of poultry, eggs, fish, and feeds.

During the surveillance period summarized in table 1, PCB's were not found in cereal grain and fresh fruits and vegetables. In late 1971, FDA established the fact that food packaging made from recycled paper which contained PCB's caused contamination of the contents, such as infant foods and cereals.

As a result of the above findings, FDA proposed certain regulations for PCB's in 1972. Table 2 shows our current regulations pertaining to PCB's in foods, animal feeds, and food packaging (ref. 1).

The first category of regulations covers the temporary tolerances for PCB's in products selected as a result of our monitoring activities, namely, milk, dairy products, poultry, eggs, animal feeds and their components, infant foods, and paper food packaging. Of these, fish are the only products which the PCB's contaminate primarily through the environment, in this case the waterways. The sources of the PCB's in the other foods and in food-packaging material were mainly industrial and agricultural uses which enabled PCB's to enter into food or animal feeds.

The second category of regulations is the prohibition of use of PCB's in such applications as heat transfer fluids, hydraulic fluids, paint components, adhesive components, etc., in plants that produce food, animal feed, or food-packaging materials.

These regulations were proposed in 1972 and are now officially established, with the exception of the maximum permissible level of PCB's in paper food-packaging material, which is an administrative guideline, pending the results of a hearing which has been granted.

Turning to the more recent period since 1972, the results obtained in FDA and USDA monitoring programs in fiscal years 1973, 1974, and 1975 are summarized in table 3. It can be seen that PCB's now contaminate far fewer types of foods than they did formerly. Although PCB's were reported in fish, milk, eggs, cheese, feed components, and poultry in fiscal years 1974 and/or 1975, the rates and levels of occurrence have declined drastically in all categories except fish.

The samples of fish FDA has analyzed were obtained through several different activities in the field. In fiscal years 1973 and 1974 we carried out Comprehensive Fish Surveys in which we analyzed freshwater and saltwater fish obtained at the wholesale level for

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Table 1. PCB findings, Nov. 1969--June 1971[a]

Food commodity	Positive findings	Avg. of positives (ppm)	Max. level (ppm)
Finfish	317	2.1	35.3
Oysters	12	Trace	Trace
Fish byproducts	6	1.8	5.0
Cheese	44	0.3[b]	1.0[b]
Milk	60	2.5[b]	22.8[b]
Eggs	17	Trace	0.5
Potato byproducts	12	1.1	4.2
Miscellaneous	11	1.9	6.5

[a] Approximately 15,000 samples examined.

[b] Fat basis.

Table 2. FDA regulations, PCB's

I. Temporary tolerances	
Commodity	PCB conc. (ppm)
Milk (fat basis)	2.5
Dairy products (fat basis)	2.5
Poultry (fat basis)	5.0
Eggs	0.5
Finished animal feed	0.2
Animal feed components	2.0
Fish (edible portion)	5.0
Infant and junior foods	0.2
Paper food-packaging material without PCB-impermeable barrier	10.0[a]
II. Use prohibited in food, feed, food packaging plants	

[a] Administrative guideline, pending hearing.

Table 3. Summary of PCB's in foods, FY '73, '74, and '75

Food Commodity	FY '73		FY '74		FY '75	
	Percent positive	Max. (ppm)[a]	Percent positive	Max. (ppm)[a]	Percent positive	Max (ppm)[a]
Fish	60.4	123.0	44.0	16.8	17.8	9.0
Milk	2.2	1.6	2.6	2.3	0.7	1.9
Eggs	1.1	Trace	4.2	11.0	0.0	N.D.
Cheese	0.9	0.5	2.6	2.8	0.0	N.D
Feed components	12.7	9.0	0.0	N.D.	0.3	0.9
Animal feeds	7.2	199.5	0.0	N.D.	0.0	N.D.
Processed fruits	4.5	19.2	0.0	N.D.	0.0	N.D.
Infant & jr. foods	1.1	Trace	0.0	N.D.	0.0	N.D.
	Percent positive	Percent above 5 ppm[a]	Percent positive	Percent above 5 ppm[a]	Percent positive	Percent above 5 ppm[a]
Meats & poultry (USDA)	1.9	0.19	1.2	0.07	0.3	0.06

[a] Milk, cheese, meats and poultry reported as ppm, fat basis.

PCB's, pesticides, and certain heavy metals. In addition, we analyzed fish obtained from our regular surveillance program for pesticides and PCB's, primarily in the raw products. Finally, we collected samples as a compliance followup after high levels of PCB's had been found in a particular species or area. The data obtained from these activities are valuable in showing which species and which areas are apt to be of concern, but it is difficult to determine whether there have been any significant trends because of the diversity of the sources of the fish samples and of the reasons for collecting them.

Figure 1 depicts the results obtained in the Comprehensive Fish Survey conducted in fiscal year 1973. It should be stressed that almost all the samples collected were commercial species intended for interstate commerce, and thus would not ordinarily include sports fish. No PCB's could be detected in over 70 percent of the samples collected. Only about 3 percent contained over 1 ppm, and 0.5 percent had over 5 ppm PCB's. It is interesting to note that the various species containing over 1 ppm were generally freshwater fish, or those which were apt to be near the shore. The only fish in this particular survey that contained more than the 5 ppm tolerance for PCB's were carp, with a maximum level of 20.5 ppm.

The results obtained in the Comprehensive Fish Survey for fiscal year 1974 are shown in figure 2. Over 80 percent of the samples analyzed in this survey did not contain PCB's, and no samples contained more than 2 ppm PCB's. However, it cannot be concluded that there was a down-trend, since it was necessary to stop this survey before it was half finished because of the necessity to divert manpower to the canned mushroom crisis. Nevertheless, freshwater fish again were most likely to contain the highest levels of PCB's.

Figure 3 depicts the results obtained with all samples of domestic finfish analyzed in fiscal years 1973, 1974, and 1975, from all FDA programs. The percentage of fish samples containing PCB's decreased in 1974 and 1975, but on the other hand, the percentage of PCB-containing samples which were above 5 ppm increased. Significantly, all these samples were from the districts in the Great Lakes area, and included such species as chubs, carp, and coho salmon.

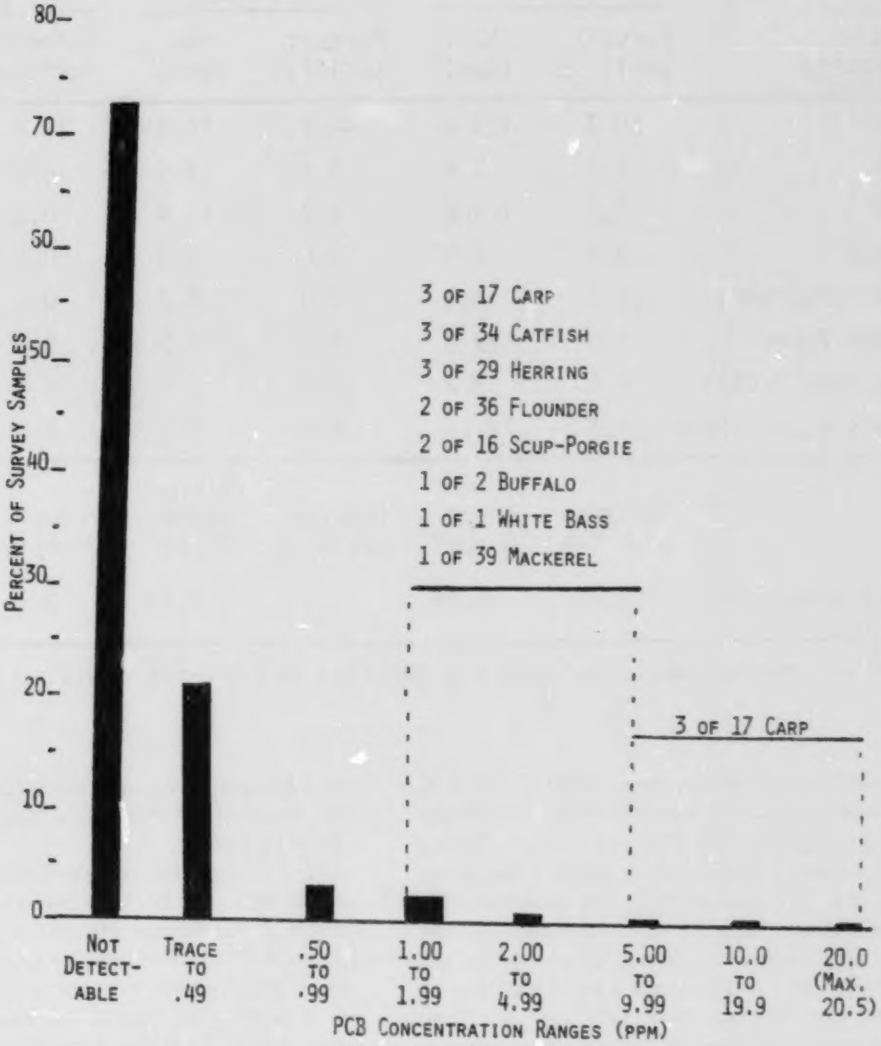

Figure 1. FY 1973 Comprehensive Fish Survey (41 species, 600 sample total).

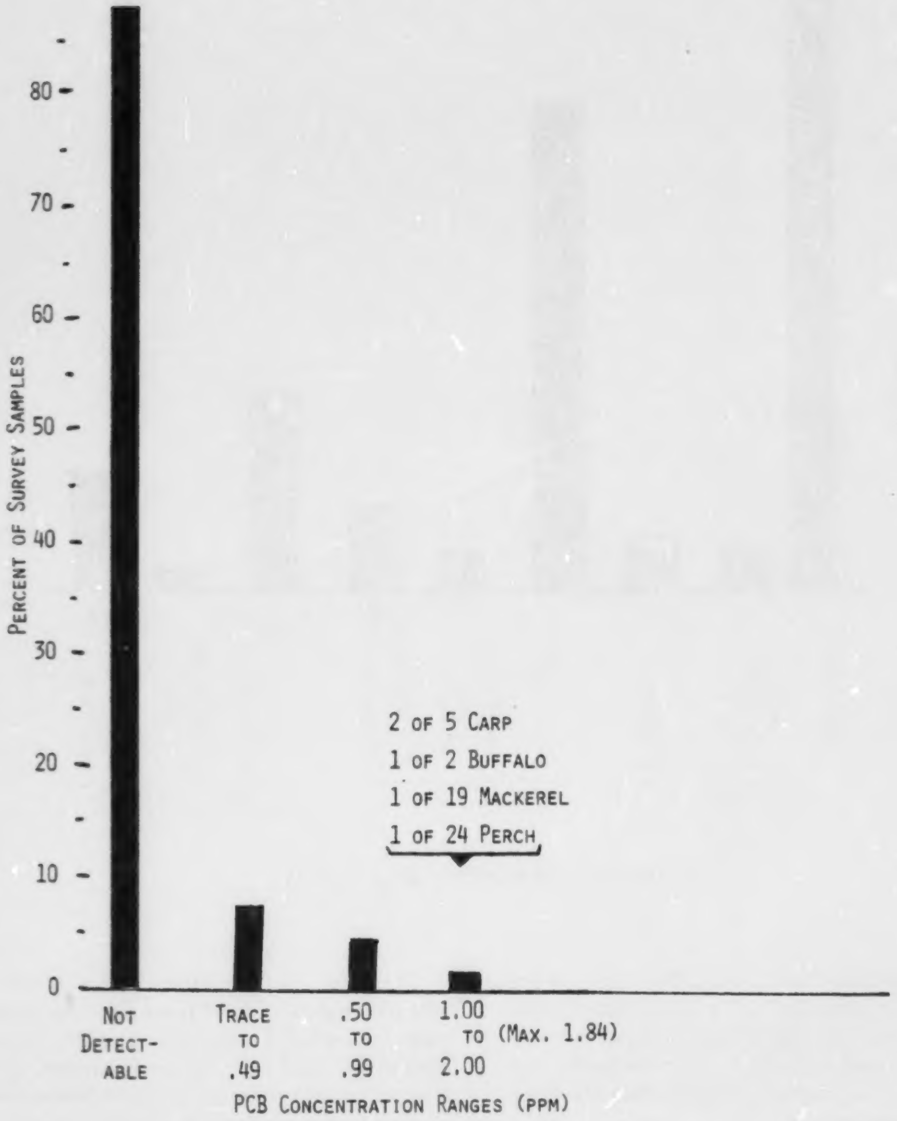

Figure 2. FY 1974 Comprehensive Fish Survey (38 species, 269 sample total).

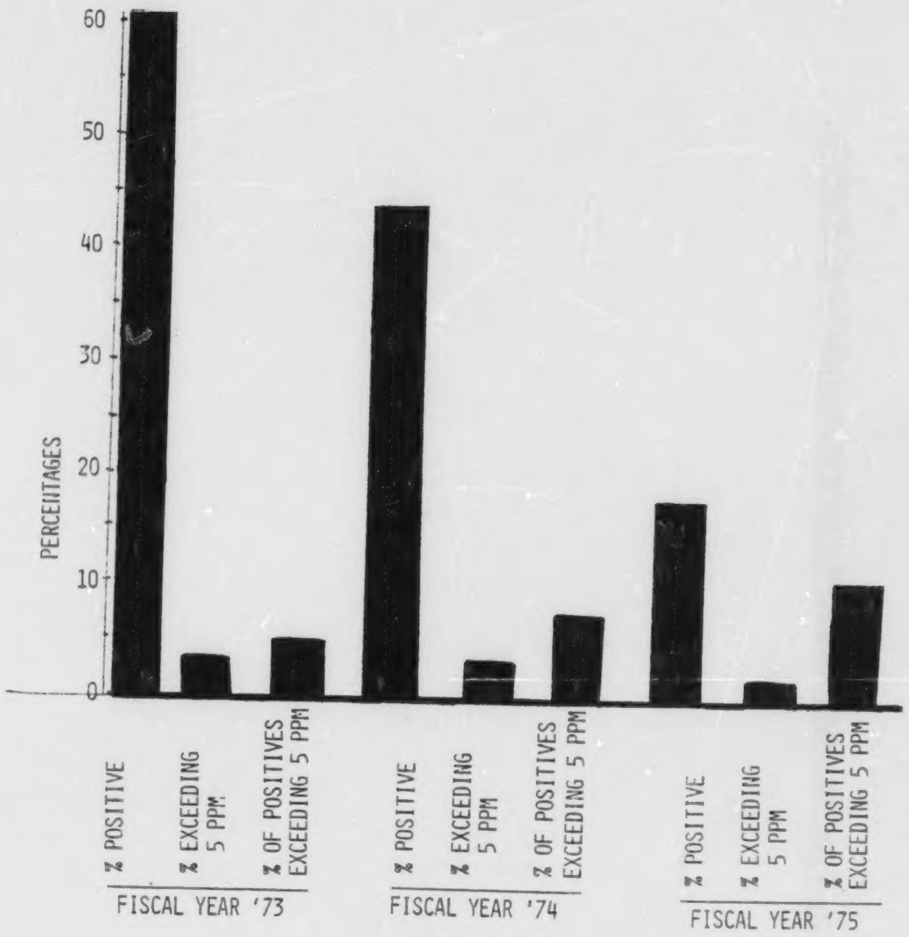

Figure 3. PCB's in fish, all programs.

As mentioned previously, it is difficult to draw any conclusions as to whether there is a significant trend, because of the changing sources and objectives from year to year. The percentage of samples examined which contained more than 5 ppm PCB's stayed in the same general range (2.2 to 3.5 percent).

These results from FDA's surveillance programs show that PCB's do not generally occur in significant levels in saltwater fish, and indeed that only a small percentage of freshwater fish in interstate commerce contain more than 5 ppm PCB's. Further, the waterways in which commercial fish are likely to contain high levels of PCB's are those which are contiguous with industrialized areas.

Thus far, we have reviewed the general levels of PCB's in individual food types. What do these findings mean in terms of average intake of PCB's from the entire diet? FDA conducts a continuing survey of the Total Diet, in which composites of 12 different food categories are analyzed. Table 4 presents the composites in which PCB's were found in the FDA Total Diet Surveys from fiscal years 1971 through the first half of fiscal year 1975. It can be seen that the meat-fish-poultry composites contained PCB's much more frequently than any other of the food groups. With regard to the findings of PCB's in the composites of fruits, vegetables, and cereals in fiscal years 1972 and 1973, it should be noted that these composites include processed and packaged

Table 4. Total Diet Studies--American teenage male

	Percent of composites containing PCB's								
	Food class composites								
Fiscal Year	Dairy products	Meat, fish & poultry	Grain & cereal products	Potatoes	Legume vegetables	Root vegetables	Garden fruits	Oils, fats & shortening	Sugars and adjuncts
1971		47	13						
1972	6	46	6		6	3	3	17	6
1973	10	33	17	3				3	
1974		43							3
1975 (1st half)		40							

Table 5. Estimates of daily PCB intakes
(Total Diet Study--teenage male)

	Average daily intake of PCB's[a]	
Fiscal year	Total diet (μg/day)	Meat-fish-poultry food class (μg/day)
1971	15.0	9.5
1972	12.6	9.1
1973	13.1	8.7
1974	8.8	8.8
1975 (1st half)	8.7	8.7

[a] Lower limit of quantitative reporting = 0.05 ppm with analytical method employed.

foods as well as raw foods purchased in retail stores. Since we have hardly ever detected PCB's in the raw products, the PCB's probably contaminated these foods during processing or from the packaging. This hypothesis is borne out by the fact that no PCB's have been found in these composites in 1974 or 1975, after our regulations had a chance to take effect.

FDA chemists have found that the source of the PCB's in the meat-fish-poultry composite is almost always due to the fish component. It is interesting to note that the frequency of findings in this composite has remained quite constant since fiscal year 1971. Significantly, PCB's are now generally found only in this fish-containing composite of the Total Diet Survey. This reflects the beneficial results of FDA and USDA in their efforts to prevent the uses of PCB's that could lead to direct contamination of foods, and of the U.S. manufacturer in limiting the sales of PCB's to "closed" electrical uses.

In calculating the average daily intake from the Total Diet Survey, we must be aware of the shortcomings in the use of composites, where the level of occurrence of the contaminant in the composites may be at or just below the limits of detection. How we handle "Trace" and "Not Detected" can have a significant effect on our estimates of the daily intake. Table 5 gives the estimated average daily intakes of PCB's by a young male adult, for fiscal years 1971 through the first half of 1975, where we have ascribed a certain level to all "Trace" in all composites and to "Not Detected" in the meat-fish-poultry composites where PCB's are still commonly detected. It can be seen that there has been a marked decrease in the estimated total intake.

However, the estimated intake will tend to continue at the FY 1975 level, as long as (1) fish remain almost the sole source of detectable PCB's and (2) the entry of PCB's into the waterways is not decreased.

In summary, the breadth of occurrence of PCB's has narrowed to the point where freshwater fish are now the primary source of PCB's in our diet. Thus, the daily PCB intake for the average citizen is low, since his consumption of freshwater fish is low, and even here, most of the commercial freshwater fish contain less than 5 ppm PCB's.

However, the estimated intake of the average consumer is only a guidepost, and the Food and Drug Administration must consider the dietary consumption patterns of significant sectors of the population which are significantly different from the average. For example, PCB intake could be quite different for those people whose diets include substantial quantities of sports fish.

For the future, (1) we must continue to monitor for PCB's in foods and food-packaging materials to maintain the beneficial results already obtained, and (2) means must be employed by the responsible Federal and State agencies to effectively halt the entry of PCB's into the aquatic environment.

REFERENCE

1. Food and Drug Administration, "Polychlorinated Biphenyls (PCB's)," *Federal Register*, Vol. 38, No. 129 (July 6, 1973), pp. 18096-18104.

LEVELS OF PCB's IN CANADIAN COMMERCIAL FISH SPECIES

John M. Graham*

Abstract

For purposes of examining PCB levels in various environments, Canada is divided into four study areas: area 1 is the Pacific sector; area 2 is Central Canada; area 3 covers the Great Lakes; and area 4 is the Atlantic sector. The tables included give detectable levels of PCB's in various species—ground fish, estuarial and pelagic fish, and crustaceans and mollusks—for the respective areas.

I will briefly summarize the work that the Inspection Branch has done in analyzing marine and freshwater species. And I am going to start by reporting on four areas in Canada (figure 1). Areas 1 and 4 involve marine species; area 2 is the central part of Canada excluding the Great Lakes; and area 3 contains the Great Lakes, which are of quite a bit of interest to us.

Table 1 is a brief summary of the PCB's that we have found in our marine species, subdivided as ground fish, pelagic, estuarial, mollusks and crustaceans, and miscellaneous. The landings are given in pounds, the levels are mean levels of PCB's, and the samples analyzed are in parentheses. One thing worth noticing is the high level in bluefin tuna, which is 2.65 ppm. We explain this by the fact that bluefin tuna is usually landed at 600 to 700 pounds. It is a very large fish and at the top of the food chain.

Another point of interest that you might note is the fish oils with a mean level of 5.1 ppm. Finally, fish meal is at a level of about 0.16 ppm. This is because, during the manufacture of fish meal, most of the oil is extracted.

As shown in figure 1, area 2 is the central part of Canada, excluding the Great Lakes. Table 2 gives PCB levels for freshwater species in that area. The production amounts to some 70 million pounds of fish annually. There are detectable levels of PCB's, but usually very, very low. These are from large northern lakes, and usually no industry is close by. The median level is 0.10 ppm.

All our samples are based on the edible portion of the fish and that portion that would normally be consumed by the consumer. The samples are taken either by commercial fishermen or by our inspectors, so that we know the exact location at which the samples are collected. Sample size is usually 15 pounds of the fillet or edible portion and a minimum of five fish.

I have subdivided the Great Lakes–St. Lawrence River system into the lakes and the river. Table 3 shows our finding on Lake Superior, which amounts to close to 3.3 million pounds of fish per year. As you can see, the lake trout is the highest in this lake, based on our findings. We also find that it varies between the inshore lake trout or shallow-water lake trout and the deepwater, or fatty, lake trout.

Table 4 shows Lake Huron, Canadian side, amounting to some 2.1 million pounds. Chub at the 2-ppm level, and carp and suckers are relatively high. Coho salmon is not normally considered a commercial species in Canada. There is quite a volume available to sports fishermen and commercial fishermen. They sell it as part of an incidental catch.

Now we come to Lake Erie (table 5), which some people say is a dead lake, but it has an annual planting of some 31 million pounds of fish. Alewives, rock bass, and carp are slightly high. The highest is catfish and again coho salmon, which is 3.14 ppm.

Table 6 shows Lake Ontario, which, as you can see, has a little bit of a problem (or a lot of problems). Smelt is at 4.16, and eels are 17.14 ppm. Again the coho salmon is up there. The eels in Lake Ontario are quite high. The eels in the St. Lawrence river system (table 7) show about 8 ppm. I have broken the eels down into their size range and given the medians.

Note the last line. A 4½-pound eel ranges from 3 to 30 ppm, so that we really cannot manage this fishery on a size or weight basis. It is going to be extremely difficult. It looks as if we are going to have to terminate the licenses for this fishery.

These eel figures are in contrast to the figures that we had for eels in the Maritime Provinces. The eels in Maritime Provinces are 0.56 ppm. So it is apparent that we will be able to keep one section of this fishery open and must try to manage or close the other one.

We presently have a 2,500-sample survey for the Great Lakes. Health & Welfare, Canada, established a temporary guideline of 2 ppm in fish; this means that we are going to have to break some of our lakes down into areas, geographic areas, and put restrictions on them either by length or weight. I do not see the problem getting any better in the next year.

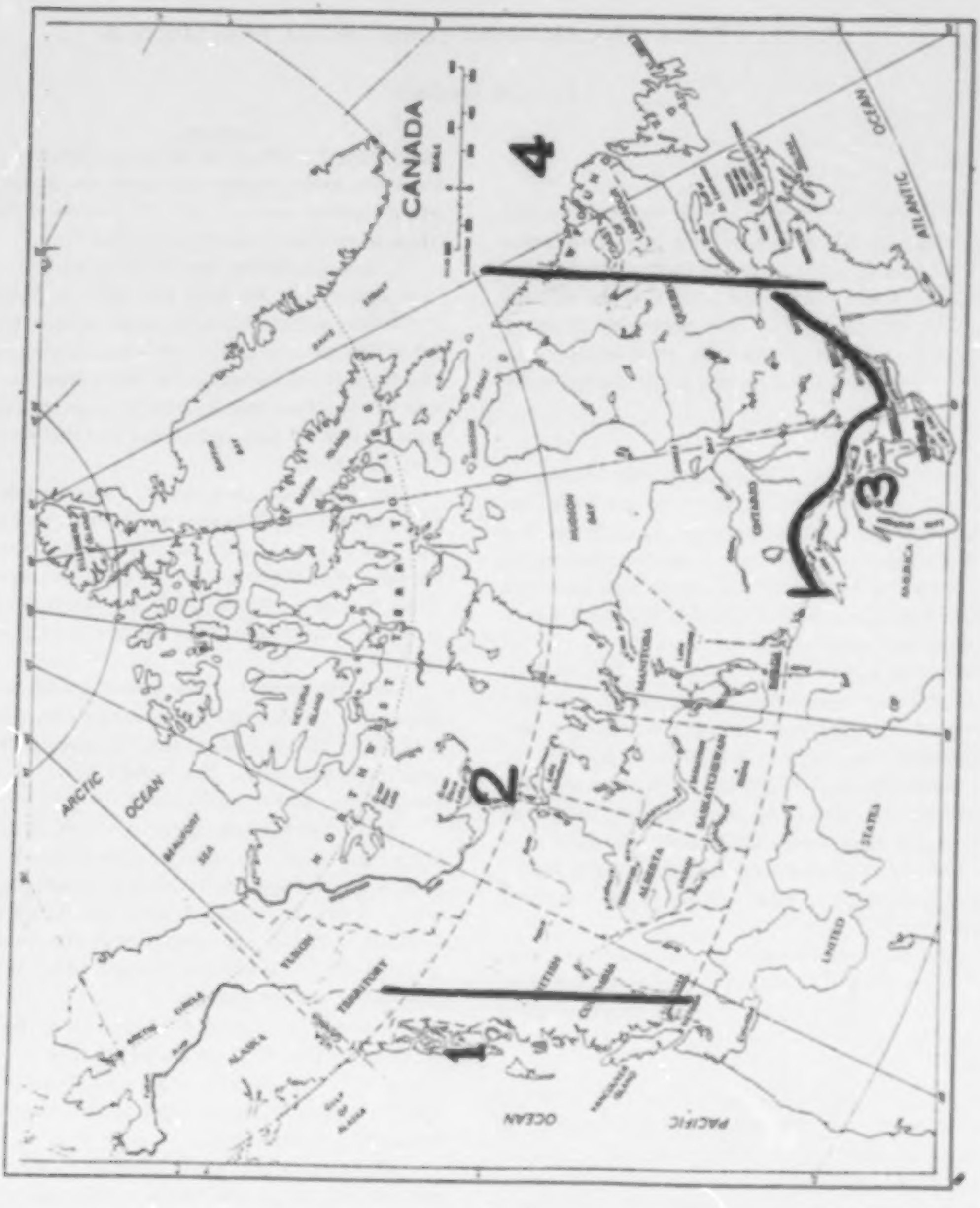

Figure 1. Four fish species areas in Canada.

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Table 1. Polychlorinated biphenyls in commercial fish and fishery products, areas 1 and 4 (marine)

	Description and species	Landings (pounds)	PCB's (mean ppm)
Groundfish	(catfish, cod, flounder, haddock, halibut, lingcod, pollock, redfish, red snapper, rockfish, sablefish, skate, sole, plaice, tomcod)	1,191,701,000	0.07 (141)
Pelagic-esturial	(Alewife; capelin; dogfish; herring; mackerel; salmon; smelt; swordfish; tuna: albacore-skipjack-yellowfin; whale)	1,131,868,000	0.39 (73)
	Bluefin	----	2.65
	Eel - Maritime Provinces	762,000	0.56 (19)
Mollusks & Crustaceans	Clams; crabs; (dungeness-queen-red-rock) lobster; mussels; oysters; scallops; shrimp	100,144,000	0.12 (56)
Miscellaneous	Fish oils	----	4.51 (12)
	Fishmeal		0.16 (71)

Table 2. Polychlorinated biphenyls in commercial fish and fishery products, area 2 (freshwater)

Species	Landings[a] (pounds)	PCB's (mean ppm)
Buffalo fish, carp, goldeye, mallet, perch, pike, pickerel, sauger, trout, whitefish	70,000,000	0.10 (113)

[a] Landings of less than 25,000 pounds per species are not included.

Table 3. Polychlorinated biphenyls in commercial fish and fishery products, (area 3)

Location	Major species	Landings[a] (pounds)	PCB (mean ppm)
Lake Superior (A)	chub	306,000	0.96 (8)
	lake herring	1,611,000	1.17 (10)
	lake trout	195,000	2.02 (37)
	whitefish	328,000	0.68 (11)
	smelt	788,000	0.35 (6)
	yellow perch	67,000	0.31 (5)
		3,295,000	

[a] Landings of less than 25,000 pounds per species are not included.

Table 4. Polychlorinated biphenyls in commercial fish and fishery products, (area 3)

Location	Major species	Landings[a] (pounds)	PCB (mean ppm)
Lake Huron (B)	chub	671,000	2.09 (17)
	whitefish	950,000	0.95 (8)
	carp	56,000	1.55 (4)
	suckers	176,000	1.33 (12)
	yellow pickerel	269,000	0.61 (9)
	sheepshead (drum)	29,000	0.75 (2)
	coho salmon	noncommercial	5.11 (6)
		2,151,000	

[a] Landings of less than 25,000 pounds per species are not included.

Table 5. Polychlorinated biphenyls in commercial fish and fishery products, (area 3)

Location	Major species	Landings[a] (pounds)	PCB (mean ppm)
Lake Erie	alewife	332,000	1.22 (14)
	rock bass	49,000	0.27 (2)
	carp	41,000	1.27 (7)
	yellow perch	12,190,000	0.19 (10)
	smelt	15,760,000	0.42 (21)
	yellow pickerel	234,000	1.16 (9)
	white bass	2,346,000	1.26 (28)
	catfish	88,000	2.04 (8)
	bullhead	109,000	0.26 (4)
	sheepshead (drum)	354,000	0.74 (15)
	coho salmon	noncommercial	3.14 (8)
		31,503,000	

[a] Landings of less than 25,000 pounds per species are not included.

Table 6. Polychlorinated biphenyls in commercial fish and fishery products, (area 3)

Location	Major species	Landings[a] (pounds)	PCB (mean ppm)
Lake Ontario (D)	bullhead	248,000	0.73 (12)
	yellow perch	699,000	1.23 (10)
	smelt	103,000	4.16 (17)
	white perch	290,000	1.84 (22)
	sunfish	203,000	0.74 (6)
	carp	395,000	1.69 (10)
	rock bass	42,000	1.76 (18)
	eel	222,000	17.14 (49)
	coho salmon	noncommercial	4.97 (3)
		2,205,000	

[a] Landings less than 25,000 pounds per species are not included.

Table 7. Polychlorinated biphenyls in commercial fish and fishery products, (area 3)

Location	Major species	Landings[a] (pounds)	PCB (mean ppm)
St. Lawrence River (E)	sturgeon	77,000	2.32 (5)
	eel	435,000	7.94 (216)

Species	Size	Range	Mean PCB
eel	<2½ pound	(0.11- 7.99)	2.95 (66)
	2½ pound-4½ pound	(1.68-22.8)	8.19 (72)
	>4½ pound	(2.82-29.7)	12.37 (43)

[a] Landings of less than 25,000 pounds per species are not included.

THE OCCURRENCE OF PCB IN THE NATIONAL FISH AND WILDLIFE MONITORING PROGRAM

Charles R. Walker*

Abstract

The National Fish and Wildlife Monitoring Program has as its objective to ascertain, on a national scale, the amounts of pesticides and contaminants in some fish, birds, and mammals. Testing is done through repeated sampling over time. The species chosen to be monitored are freshwater fish, mallard and black duck, and starling.

PCB residues in freshwater fish were found to be highest in certain river systems that lie in industrial areas. For mallards and black ducks, concentrations were greatest in ducks in the Atlantic flyways. Starlings from all over the Nation showed PCB contamination; however, over the 1970-1974 period, there was an apparent decline in residue levels, except in certain specific areas such as Austin, Alabama. Related monitoring indicates that PCB may be especially threatening to endangered species, with serious effects on the survival of their young.

The National Fish and Wildlife Monitoring Program was initiated in 1967 as a cooperative Federal effort conducted jointly by the Bureau of Commercial Fisheries and the Bureau of Sport Fisheries and Wildlife in the U.S. Department of the Interior, and the Water Supply and Sea Resources Program of the National Center for Urban and Industrial Health, Public Health Service, U.S. Department of Health, Education, and Welfare (ref. 34). The current National Pesticide Monitoring Program is sponsored by the Monitoring Panel of the Federal Working Group on Pest Management that includes components drawn from the Environmental Protection Agency, the Tennessee Valley Authority, the National Science Foundation, and the Departments of Agriculture, Defense, Commerce, Interior, and Health.

The monitoring objective was to ascertain on a national scale the amount of pesticides and related materials in components of the environment and trends of these levels with time through repeated sampling and analysis of environmental components (refs. 34,37,38). The Fish and Wildlife Service program consists of three elements: (1) freshwater fish, (2) mallard and black duck, and (3) starlings.

The Freshwater Fish Program originally consisted of sampling fish from 50 stations in the continental United States as it was initiated in 1967 (refs. 24,25). In 1970 this was expanded to include 100 stations sampled on an annual basis and included the conterminous United States and Alaska (ref. 30). Criteria for selection of specific sampling station locations included the following, in the order of priority: (1) availability of desired species of fish; (2) relationship to station locations for other phases of the National Pesticide Monitoring Program (refs. 11,14,19,67,69), particularly those stations used for monitoring surface water samples; (3) existence of State-sponsored monitoring programs or cooperative participation by a State conservation agency; and (4) availability of suitable manpower and facilities to make field collections. In the late 1960's, the toxicological significance and the detection of polychlorinated biphenyl compounds in fish and wildlife along with the interferences with organochlorine pesticide analysis became apparent (refs. 31,32,33,43,45,50,54,66). This problem stimulated the development of analytical methods for the separation and identification of PCB in fish samples from the National Monitoring Program (refs. 26,28,52,53,57,58).

The cleanup of solvents (refs. 20,56), selective partition of PCB from pesticides (refs. 1,3,4,7,16,18,26,53,55,58,59), quantitative determination (refs. 2,9,46,54,57,62), and confirmation methods (refs. 2,6,8,29,51,52) utilizing gas chromatography-mass spectrometry became practical for Federal agencies to use in the National Pesticide Monitoring Program by 1970. In the Freshwater Fish Program, samples of three to five fish were taken of three species representing different tropic levels to comprise each composite sample. All composite samples of white fish were analyzed for the most common organochlorine insecticides, including DDT, DDE, TDE, dieldrin, aldrin, endrin, BHC, heptachlor epoxide, chlordane, toxaphene, and beginning with the 1970 collections, polychlorinated biphenyl compounds (PCB), as well as the percent lipid content of the fish (ref. 61). Whereas these analysis were contracted to private and other Federal laboratories, a cross-check quality assurance program was conducted on about 10 percent of the samples by our Fish-Pesticide Research Laboratory at Columbia, Missouri. The number of pesticides and related contaminates that were detected in the cross-check analyses grew from 19 compounds in 1970 to 33 parent compounds, homologs, or metabolites by 1973 (table 1).

The mallard and black duck monitoring program

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Table 1. Organochlorine pollutants detected by initial and confirmatory laboratories from 1967-1974

Pollutant	1967	1968	1969	1970	1971	1972	1973
DDE	I[a]	I	I	I C[b]	I C	I C	I C
DDD o,p'isomers	I	I	I	I C	I C	I C	I C
DDT	I	I	I	I C	I C	I C	I C
DDE				C	C	C	C
DDD p,p'isomers				C	C	C	C
DDT				C	C	C	C
1232				C	O[c]	O	O
1242					C	O	O
1248 Aroclor^{R}				C	C	C	O
1254			I	I C	I C	I C	I O
1260				C	C	C	I O
dieldrin	I	I	I	I C	I C	I C	I C
aldrin	I	I	O	I C	I C	O O	I C
endrin	I	I	O	I C	I C	I C	I C
BHC			I	I C	I C	I C	I C
lindane	I	I	O	O	O	O	O C
heptachlor	I	I	I	I C	O C	O C	O C
heptachlor expoide	I	I	I	I C	I C	I C	C
chlordane	I	I	I	O C	I C	I O	C
cis-chlordane					C	C	C
toxaphene	I	I	O	O C	I C	I C	I C
hexachlorbenzene (HCB)					C	C	C
heptachlor norborene							C
oxychlordane							C
hexachlor norbornadiene							C
2,4-D PGBE							C
pentachlorobenzene							C
hexachloro-1,3-butadiene							C
ethylhexylphthalate				C	O	O	O
dibutylphthalate					C	O	O
dioctylphthalate					C	O	O
DEHP						C	O

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[a] I-indicates a pollutant was reported in the results from the initial analyses.

[b] C-indicates a pollutant was reported in the results from the confirmatory analyses.

[c] O-indicates a pollutant was found in previous years but not reported later.

was conducted in cooperation with State agencies in conjunction with the fall migration of waterfowl and the hunting season in each of the conterminous States (refs. 17,21,22,23,64). Through a process of systematic subsampling, a series of wings, approximately 12,500 annually, are drawn from each State. These are composited for analysis into samples of 25 wings each representing a sample from each State in proportion to the harvest. Mallard duck wings are collected in the Pacific, Central, and Mississippi flyways whereas both the mallard and black duck were represented in those samples from the Atlantic flyway, depending on the availability of species. Pesticide analysis included chlorinated hydrocarbon pesticides and beginning with the 1969 sampling also included polychlorinated biphenyl compounds.

The National Monitoring Program for starlings, a widely distributed and abundant species, was especially designed to sample five degree latitude/longitude blocks covering the contiguous 48 States with up to four randomly selected collecting sites within each block (refs. 34,41,42,63). Ten birds were collected at each sample site and composited for analysis in the fall of even years at approximately 125-130 sites. Organochlorine pesticides were analyzed in the 1967-68 samples and beginning with the 1970 samples polychlorinated biphenyl compounds were analyzed in 1970-72 and 1974 samples.

Freshwater Fish Monitoring Program

Polychlorinated biphenyl compounds have been detected in certain rivers and areas in unusually high concentrations and confirmed in a quality control cross-check analysis among several laboratories (refs. 54,61).

PCB residues have been found in 40 to 60 percent of the samples in residue concentrations exceeding 0.5 mg/kg (figure 1). More than 98 percent of the samples during the period of 1969-71 contained residues in at least one composite sample exceeding these criteria. Geographically, the higher concentrations appear to be

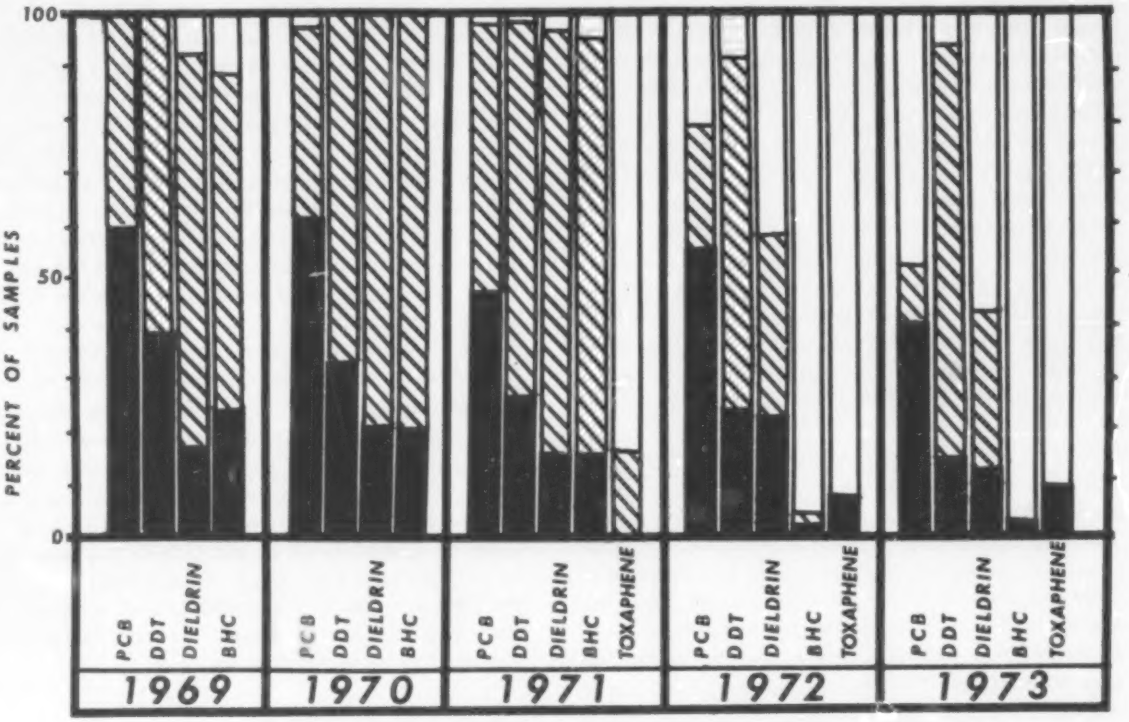

Figure 1. Percentage of fish sampled from 1969-1973 in the National Pesticide Monitoring Program that contained either no detectable residues (white), residues (cross-hatched), or levels exceeding the criteria established to be biologically significant (solid black: >0.5 mg/kg PCB: >1.0 mg/kg DDT; and >0.1 mg/kg dieldrin, BHC, or toxaphene) (ref. 69).

associated with certain river systems having industrial activity (refs. 54,61) (tables 2 and 3). PCB residues expressed as Aroclor 1254 were found in five major river systems in the Atlantic coastal region, with residues exceeding 5 mg/kg. Four of these stations had residues exceeding 10 mg/kg during the last 5 years. Fish in four of the Great Lakes stations had PCB concentrations exceeding the 5 mg/kg level and all stations reported concentrations exceeding 0.5 mg/kg. In the Mississippi River system, the Allegheny and Ohio were the hot spots, with seven out of the eight stations reporting residue concentrations in excess of 5 mg/kg. Thirty-one of thirty-five stations in this river system reported residues in excess of 0.5 mg/kg in the 1970-73 sampling programs. The highest residues, often exceeding 10 mg/kg were found in the Allegheny, Kanasha, Cumberland, Tennessee, and Ohio Rivers along with stations on the Mississippi River at Memphis, Tennessee, and the Missouri River at Herman, Missouri. Other monitoring stations that were found to have residue levels exceeding 5 mg/kg during the sampling periods 1970-73 included: the Williamette River on the Columbia system; the Rouge River in the Pacific coastal drainage; the Sacramento River in California; the Chena River tributary of the Yukon in Alaska; and the Rio Grande, Alabama, and Mississippi Rivers in the Gulf States region. Only in two sample periods of 1972-73 and in the current monitoring samples, which are still yet to be fully analyzed, has there been a downward trend, but this occurs only in those samples where residues are not being detected. The stations where high residues have been noted in the past still remain relatively contaminated with PCB. Unlike the decline of DDT in Great Lakes fishes, PCB concentrations do not show significant changes and may trend upward in salmonids, as indicated in special investigations conducted by Willford (ref. 69), and Veith (ref. 60).

Just as it is exceedingly important to distinguish PCB from organochlorine pesticides, we must also make positive identifications of the different Aroclors so that they may be correctly expressed in stoichiometric calculation. For example, in table 4, let us examine some of those stations with high PCB concentrations. The composition of PCB residues in selected fish samples taken during the National Pesticide Residue Monitoring Program illustrates the difficulties in equating all residues to one type of Aroclor, such as 1254. In this illustration we can readily show that the expression of PCB from the Ohio, Mississippi, and Hudson drainage would certainly misrepresent the true distribution of PCB isomers if they were all stoichiometrically expressed as 1254. The best analogy I can offer to illustrate this is if we were to express all of the hardness or alkalinity of water as calcium carbonate, when indeed the actual chemical analysis indicated that most of the carbonates existed as magnesium carbonate.

This relationship has two dimensions. The first is that the lower Aroclor series are more highly biodegradable than are the higher series such as 1254 and 1260. A PCB residue in fish taken downstream following degradation would thus appear to be composed primarily of the 1254 or 1260 isomer. However, the toxicity and potential biological effect would be more serious with regard to the Aroclors 1232, 1242, and 1248, as noted in table 4. This was particularly evident in the 1970 cross-checks, in which we were finding that results of two other laboratories typically expressed PCB levels at concentrations of only 5 to 20 percent of that found in the sample by the more sophisticated procedures used by our Fish-Pesticide Research Laboratory (refs. 52,54,55,57,58).

Mallard and Black Duck Monitoring Program

White and Heath report that polychlorinated biphenyl residues were highest in the Atlantic flyways; the mean value of 1.36 ppm occurred in black duck in both 1969 and 1972 (ref. 64). Mallards from the same flyway averaged 1.24 and 1.29 ppm for samples taken in 1972 and 1969 respectively (figure 2 and table 5). These levels compared to mallard duck wings from the Mississippi flyway, where average values were found to be 0.66 and 0.44 ppm on a wet weight basis for the years 1972 and 1969. Both the Central and Pacific flyways had residues of approximately 0.1 ppm during 1972 and 0.2 ppm in 1969. The overall trend in the concentration of PCB would appear to be downward in the Central and Pacific flyways; however, this was significantly different at the $P < 0.05$ in the analysis of variance calculation. The increase in concentration observed in the Mississippi flyway and status quo for the Atlantic flyway have not been significantly changed over the same period of time (refs. 21,22,23,64). The recent studies by White and Kaiser (ref. 65) on the mortality of ruddy ducks in the Delaware system, however, did indicate the seriousness of PCB residues. They found Aroclor 1260 residues or the bioaccumulation of pentachloro and hexachloro biphenyl compounds that exceed the levels found in our National Monitoring Program by almost tenfold.

Starling Monitoring Program

PCB has been found in all of the starling samples taken in 1970, 1972, and 1974 (figure 3). The level of residues appears to be declining throughout this period; however, some areas continue to report exceedingly high concentrations—for example, in the Austin, Alabama,

Table 2. Stations with residues in at least one composite exceeding 0.5 mg/kg total PCB's, from an estimate based on Aroclor 1254 (asterisk and double asterisks denote residues in excess of 5.0 and 10.0 mg/kg, respectively)

River (location)	1970	1971	1972	1973
Atlantic Coastal Streams				
Penobscot R. (Old Town, ME)			X	
(Stillwater R.)				
Kennebec R. (Hinckley, ME)				
L. Champlain (Burlington, VT)	X	X	X	X
Merrimac R. (Lowell, MA)	X*	X**	X**	X**
Connecticut R. (Windsor Locks, CT)	X*	X**	X**	X*
Hudson R. (Poughkeepsie, NY)	X**	X**	X**	X**
Raritan R. (Highland Park, NJ)	X*	X	X*	X
Delaware R. (Camden, NJ)	X	X	X**	X**
Susquehanna R. (Conowingo, MD)	X	X	X	X
Potomac R. (Little Falls, MD)	X	X	X	X
James R. (Richmond, VA)	X	X		X
Roanoke R. (Roanoke Rapids, NC)	X	X	X	X
Cape Fear R. (Elizabethtown, NC)	X	X	X	
Pee Dee R. (Dongola, SC)	X		X	X
Cooper R. (Summerton, SC)	X		X	X
Savannah R. (Savannah, GA)	X		X	X
Altamaha R. (Doctortown, GA)	X		X	
St. Johns R. (Welaka, FL)	X	X		X
St. Lucie Canal (Indiantown, FL)	X			
Gulf Coastal Streams				
Suwanee R. (Old Town, FL)	X		X	
Apalachicola R. (Jim Woodruff Dam, FL)	X	X	X	X
Alabama R. (Chrysler, AL)	X	X	X*	X
Tombigbee R. (McIntosh, AL)	X	X		
Mississippi R. (Luling, LA)	X	X	X*	X
Brazos R. (Richmond, TX)	X	X	X	X
Colorado R. (Wharton, TX)	X	X	X	X
Nueces R. (Mathis, TX)				

Table 2. (con.)

River (location)	1970	1971	1972	1973
Rio Grande (Brownsville, TX)				X*
Rio Grande (Elephant Butte, NM)	X	X	X	
Rio Grande (Alamosa, CO)	X*			X
Pecos R. (Red Bluff Lake, TX)	X		X	
Great Lakes Drainage				
Gennessee R. (Scotsville, NY)	X	X	X	X
St. Lawrence R. (Massena, NY)	X*	X*	X**	X
L. Ontario (Port Ontario, NY)	X	X**	X*	X*
L. Erie (Erie, PA)	X	X	X	
L. Huron (Bay Port, MI)	X	X*	X	X*
L. Mighigan (Sheboygan, WI)	X	X**	X*	X*
L. Superior (Bayfield, WI)	X	X	X	X
Mississippi River System				
Allegheny R. (Natrona, PA)	X**	X**	X**	X*
Kanawha R. (Winfield, WV)	X**	X	X**	X*
Wabash R. (New Harmony, IN)	X	X	X	X
Ohio R. (Marietta, OH)	X**	X**	X**	X**
Ohio R. (Cincinnati, OH)	X**	X**	X**	X**
Ohio R. (Metropolis, IL)	X	X*	X*	X
Cumberland R. (Clarksville, TN)	X	X	X**	X*
Tennessee R. (Savannah, TN)	X	X	X**	X*
Wisconsin R. (Woodman, WI)	X	X	X	X
Des Moines R. (Keosauqua, IA)	X	X	X	X
Illinois R. (Beardstown, IL)	X	X	X	X
Mississippi R. (Little Falls, MN)	X			
Mississippi R. (Guttenburg, IA)	X	X	X	X
Mississippi R. (Cape Girardeau, MO)	X	X	X	X
Mississippi R. (Memphis, TN)	X	X	X**	X**
Arkansas R. (Pine Bluff AR)	X	X		
Arkansas R. (Keystone Reservoir, OK)	X	X	X	
Arkansas R. (John Martin Reservoir, CO)				
Verdigris R. (Oologah, OK)	X			
Canadian R. (Eufaula, OK)	X			

Table 2. (con.)

River (location)	1970	1971	1972	1973
White R. (DeValls Bluff, AR)	X	X	X	
Yazoo R. (Redwood, MS)	X	X		
Red R. (Alexandria, LA)	X	X		X
Red R. (Lake Texoma, OK)				
Missouri R. (Hermann, MO)	X	X**	X*	X*
Missouri R. (Nebraska City, NE)	X	X	X	X
Missouri R. (Garrison Dam, ND)				
Missouri R. (Great Falls, MT)	X	X	X	X
Big Horn R. (Hardin, MT)	X		X	
Yellowstone R. (Sidney, MT)				
James R. (Olivet, SD)	X	X	X*	X
N. Platte R. (Lake McConaughy, NE)				
S. Platte R. (Brule, NE)		X	X	X
Platte R. (Louisville, NE)	X		X	X
Kansas R. (Bonner Springs, KS)	X	X	X	X
Hudson Bay Drainage				
Red R. (North) (Noyes, MN)	X	X	X	X
Colorado River System				
Green R. (Vernal, UT)				
Colorado R. (Imperial Reservoir, AZ)				
Colorado R. (Lake Havasu, AZ)				
Colorado R. (Lake Mead, NV)		X	X	
Colorado R. (Lake Powell, AZ)				
Gila R. (San Carlos Reservoir AZ)				
Interior Basins				
Truckee R. (Fernley, NV)	X	X	X	X
Utah L. (Provo, UT)		X		
Bear R. (Preston, ID)	X	X	X	X

Table 2. (con.)

River (location)	1970	1971	1972	1973
California Streams				
Sacramento R. (Sacramento, CA)	X	X	X*	X
San Joaquin R. (Los Banos, CA)	X		X	
Columbia River System				
Salmon R. (Riggins, ID)	X	X		
Snake R. (Hagermann, ID)	X	X	X	X
Snake R. (Lewiston, ID)	X	X	X	
Sanke R. (Ice Harbor, WA)	X	X	X	X
Yakima R. (Granger, WA)	X		X	
Williamette R. (Oregon City, OR)	X	X	X*	X
Columbia R. (Bonneville, OR)	X		X	X
Columbia R. (Pasco, WA)	X	X	X	X
Columbia R. (Grand Coulee, WA)	X	X	X	X
Pacific Coastal Streams				
Klamath R. (Hornbrook CA)				
Rogue R. (Gold Ray Dam, OR)	X	X	X	X*
Alaskan Streams				
Chena R. (Fairbanks, AK)	X	X	X*	X
Kenai R. (Soldatna, AK)				
Hawaiian Streams				
Waikele Stream (Waipahu, HI)	X	X	X	X
Manoa Stream (Honolulu, HI)			X	

Table 3. Environmental contaminants in fish (refs. 52, 53, 54, 55)

	Residue (μg/g whole body)[a]			
	1970	1971	1972	1973
Hudson, R.,				
Poughkeepsie,		93	61	104
N.Y.	213	76	118	69
Ohio R.				
Marietta,	122	77	16	7.5
Ohio	38	-		6.0
Cincinnati,	133	156	20	45
Ohio	66	-	43	
Metropolis,				8.6
Ill.				
Lake Michigan				
Sheboygan,	7.8		2.6	11
Wis.		53	4.9	4.4
Merrimac R.				
Lowell,				
Maine	98	45	8.8	8.5

[a] Each value represents analysis of single fish.

Table 4. Composition of PCB residues in selected fish samples from the 1970 National Pesticide Residue Monitoring Program (ref. 54)

River	Location	Species	PCB residue as Aroclor type (μg/g whole body)				
			1232	1248	1254	1260	total
Ohio	Cincinnati, O.	Carp	10.2	75	42	6.0	133
Ohio	Cincinnati, O.	White crappie	15.9	17	27	5.6	66
Ohio	Marietta, O.	Channel catfish	38	23	11	4.9	77
Ohio	Marietta, O.	Channel catfish	15.6	5.2	12.6	4.6	38
Yazoo	Redwood, Miss.	Smallmouth buffalo	72	—	1.4	—	73
Hudson	Poughkeepsie, N.Y.	Goldfish	8.6	173	32	—	213
Allegheny	Natrona, Pa.	Walleye	—	5.2	25	4.6	35
Delaware	Camden, N.J.	White perch	—	8.0	6.8	3.9	19
Cape Fear	Elizabeth Town, N.C.	Gizzard shad	18.9	—	2.6	1.1	23
Lake Ontario	Port Ontario, N.Y.	White perch	12.9	—	4.6	1.2	19
Mississippi	Memphis, Tenn.	Drum	11.2	—	4.5	3.4	19
Merrimac	Lowell, Mass.	Yellow perch	13.8	75	6.1	3.2	98

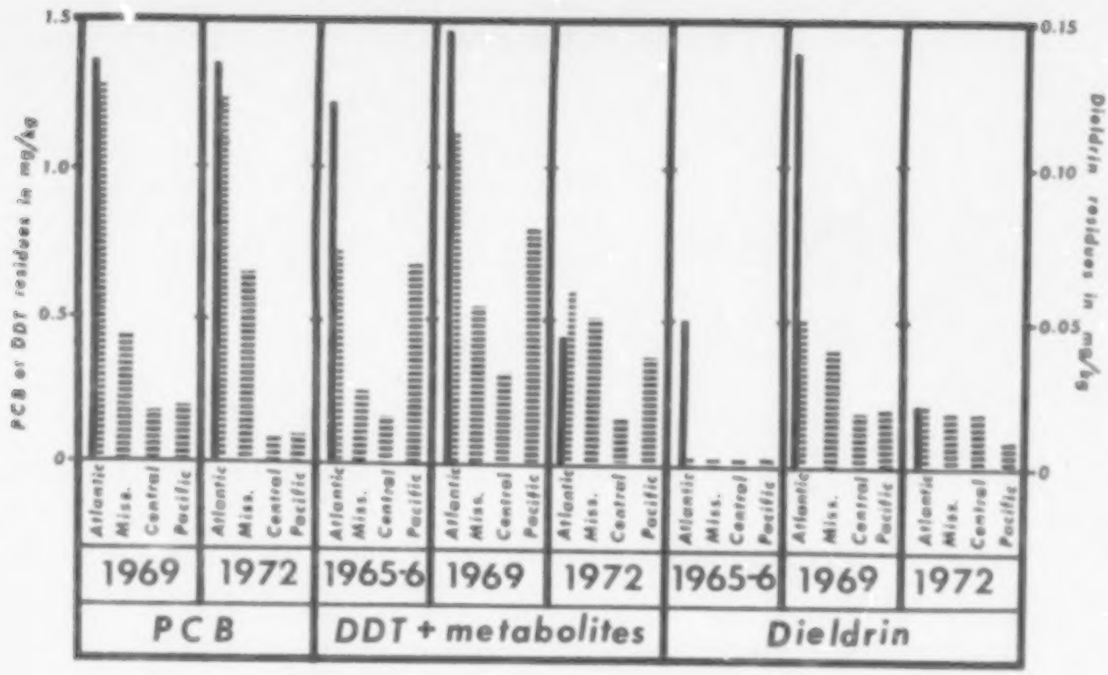

Figure 2. Trends of PCB and organochlorine pesticide residues measured in samples of duckwings from each flyway in the National Pesticide Monitoring Program: black duck (solid bar); mallard (broken bar).

station, the 1970 analysis showed 24.3 ppm in 1972 and 19.9 ppm and a 1.8 ppm concentration in 1974 (table 6). PCB's appear to be ubiquitous throughout the Nation and occur generally in a concentration of less than 5 ppm, with an arithmetic mean for 1970 of 0.65 ppm, for 1972 of 0.43 ppm, and for 1974 of 0.12 ppm on the wet weight basis (figure 4).

Related Investigative Monitoring Programs for Wildlife

The widespread occurence of PCB in ecosystems may be most serious to fish-eating species (refs. 5,10,13) and to the rates of bioaccumulation up the food chain (refs. 38,43,49). Special investigative monitoring activities of the U.S. Fish and Wildlife Service laboratories at Patuxent, Maryland, have been reported on the bald eagle relative to pesticides and PCB levels vs. autopsy and analysis (refs. 15,40,44). In endangered or threatened species, PCB residues may have serious effects on survival of young since some effects have been documented on certain behavioral patterns (refs. 35,36). Residues observed in the National Monitoring Program may pose a hazard to animals that have demonstrated particular sensitivity to PCB concentrations affecting organ function, behavior, growth, and/or reproduction (refs. 27,36,48,50,52).

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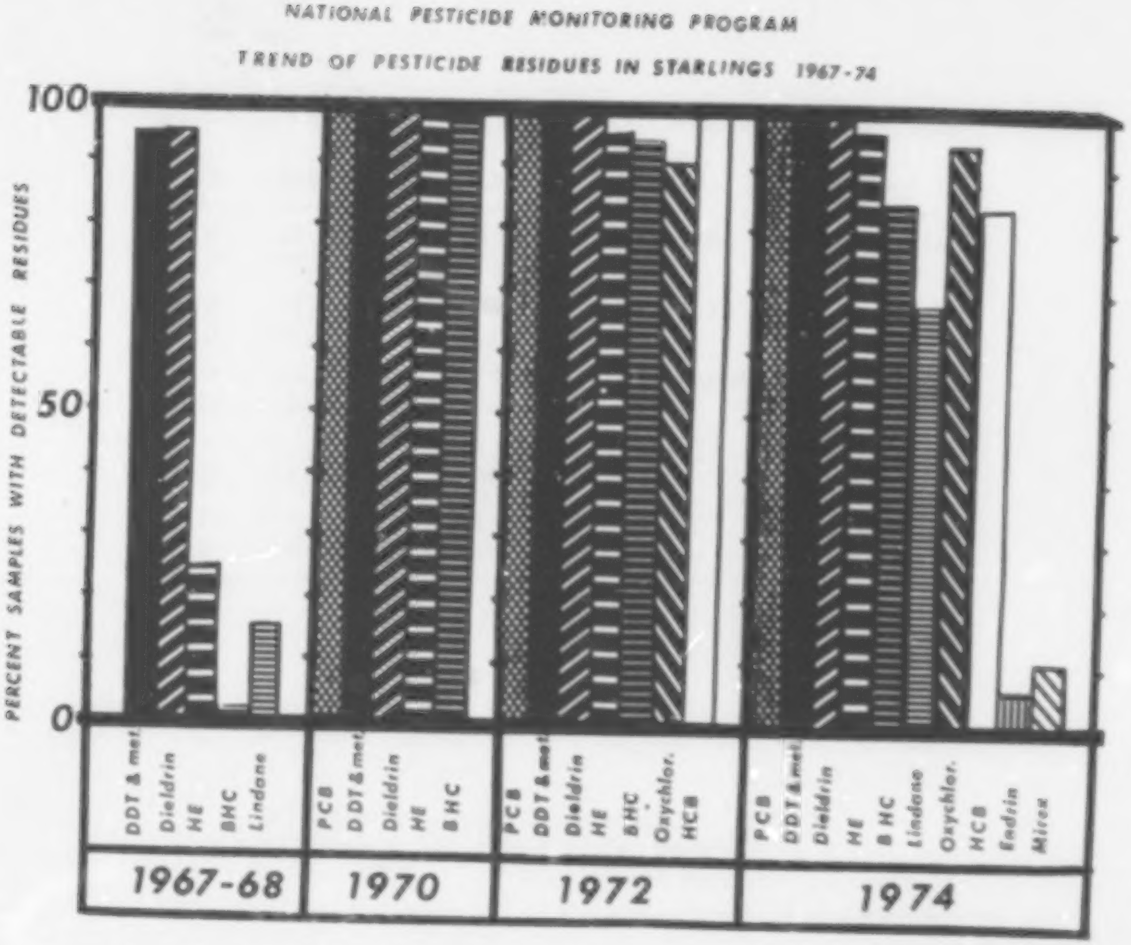

Figure 3. Percentage of starling samples containing PCB and organochlorine pesticides in the National Pesticide Monitoring Program.

Table 5. Mean residues of PCB in wing-pools by major flyway, 1972 and 1969

Species	Flyway	Year	No. of Pools	Residues (ppm wet weight) Mean	Residues (ppm wet weight) Std. Error
Black Duck	Atlantic	1972	44	1.36	0.149
		1969	42	1.37	0.161
Mallard	Atlantic	1972	21	1.24	0.230
		1969	19	1.29	0.457
Mallard	Mississippi	1972	61	0.66	0.303
		1969	51	0.44	0.061
Mallard	Central	1972	56	0.10[a]	0.013
		1969	49	0.20	0.039
Mallard	Pacific	1972	55	0.11[a]	0.009
		1969	51	0.20	0.014

[a] $P < 0.05$ (Analysis of variance).

Table 6. Means, ranges, and geometric mean PCB in starlings from each collecting period, 1967-68 through 1974

Year	No. of Pools	PCB[b] (ppm wet weight) $\bar{x} \pm$ S.E.	PCB[b] (ppm wet weight) Range	PCB[b] (ppm wet weight) Geom. $\bar{x}$
1967-68	360	-	-	-
1970	125	0.663 ± 0.196	0.09 - 24.30	0.358
1972	130	0.425 ± 0.153	0.037 - 19.90	0.215
1974***[a]	126	0.112 ± 0.016	0.006 - 1.88	0.068

[a] Mean residues for 1974 were significantly lower than for 1972:

*** = $P<0.001$.

[b] PCB were not analyzed in 1967-68.

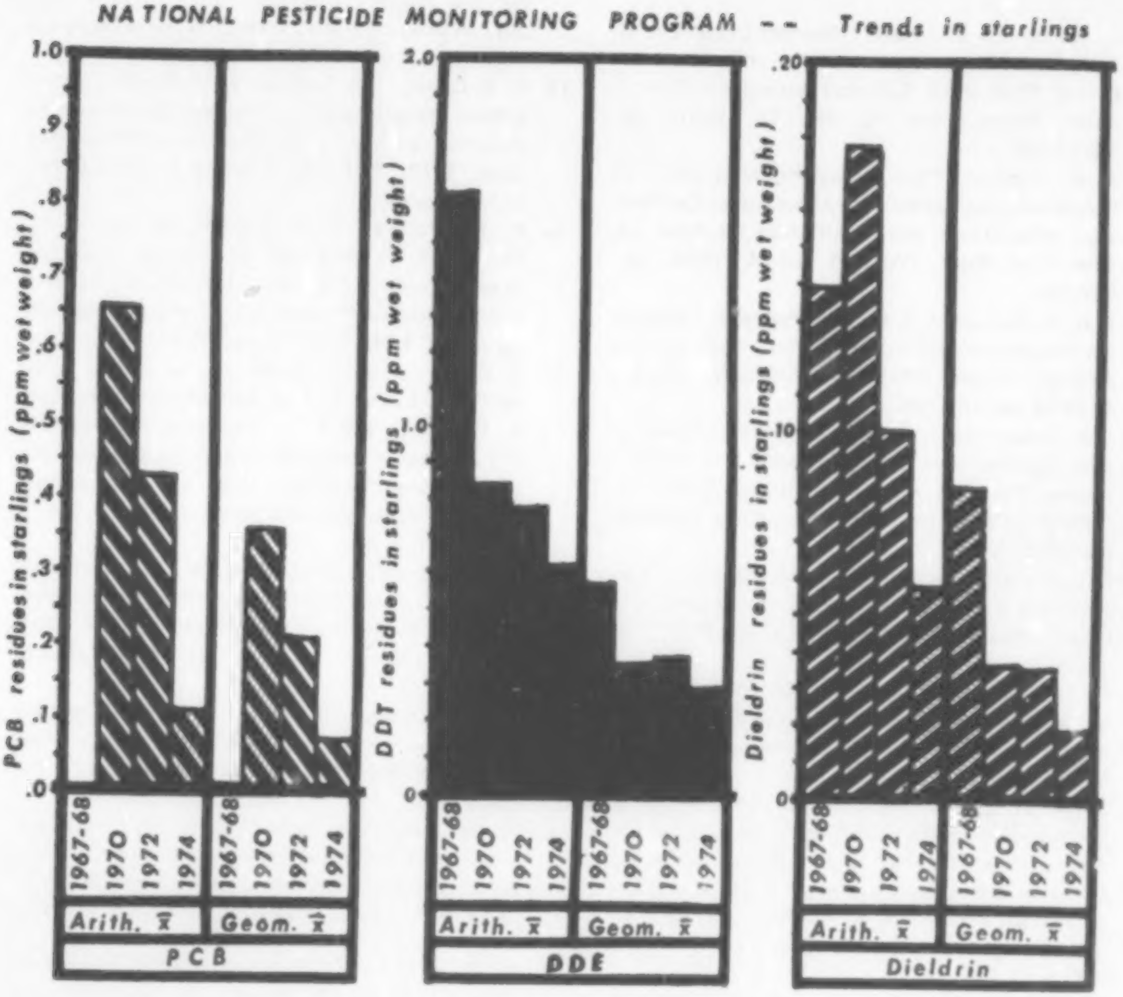

Figure 4. Trends of PCB and organochlorine pesticide residues measured in pooled samples of starlings taken in the National Pesticide Monitoring Program.

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TRENDS OF POLYCHLORINATED BIPHENYLS IN THREE LAKE MICHIGAN FISHES

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and Lawrence W. Nicholson†

Abstract

*Concentrations of polychlorinated biphenyls (PCB's) were determined from 1972 through 1974 in three species of fish collected in the fall from eastern Lake Michigan. Mean concentrations in whole fish ranged in different years from 5.24 to 5.66 ppm in bloaters (*Coregonus hoyi*), 10.4 to 12.2 ppm in coho salmon (*Oncorhynchus kisutch*), and 12.9 to 22.9 ppm in lake trout (*Salvelinus namaycush*). There was no evidence of a decline in average PCB residues in the fish sampled after the introduction in 1970-71 of voluntary controls on the sale of PCB's in the United States.*

INTRODUCTION

Past investigations have shown that substantial levels of polychlorinated biphenyls (PCB's) were present in fish from the open waters of Lake Michigan during the late 1960's. Veith and Lee (ref. 1) reported residues of Aroclor 1254 ranging from 14 to 25 ppm in three coho salmon (*Oncorhynchus kisutch*) and 6 to 25 ppm in eight rainbow trout (*Salmo gairdneri*) collected from western Lake Michigan during the spring of 1969. Stalling and Mayer (ref. 2) reported a PCB concentration (sum of Aroclors 1248 and 1254) of 13 ppm in a single coho salmon collected from eastern Lake Michigan during the fall of 1969. Extensive collections and analyses of fish from Lake Michigan in 1971 confirmed the approximate levels of PCB contamination found in 1969, and further demonstrated that residues in excess of 5.0 ppm were common in several species of fish (refs. 3,4).

Because of the potential effects of PCB's on fish and consumers of fish, the Great Lakes Fishery Laboratory in 1972 incorporated analysis of PCB's with its routine program of pesticide analysis in Lake Michigan fishes. The program is designed to permit the accurate determination and statistical evaluation of contaminant levels and trends with time in fish from Lake Michigan. Particular emphasis is placed on the yearly evaluation of contaminants in samples of bloaters (*Coregonus hoyi*) that are similar in terms of fish size, location of catch, and time of year sampled. Bloaters are particularly useful for contaminant evaluation for several reasons: they represent an important commercial species that concentrates pesticides and PCB's; they reflect local conditions because they are essentially nonmigratory; and they are generally easy to catch in adequate numbers. Lake trout (*Salvelinus namaycush*) and coho salmon are also sampled annually because of their current importance as sport fishes and their role in salmonid restoration programs. In addition, the analysis of lake trout provides an estimate of contaminant levels in a long-lived predatory species from the same geographic location as the bloaters; and coho salmon serve as potential indicators of lakewide contaminant trends because of their extensive migrations.

This report presents the trends of average PCB concentrations in Lake Michigan bloaters, lake trout, and coho salmon in 1972-74. The data provide a basis for determining the effectiveness of past efforts to reduce the contribution of PCB's to the Lake Michigan environment, as evidenced by the level of contamination in the fisheries. Voluntary restrictions on PCB sales in the United States were introduced in 1970-71.

FISH SAMPLING PROCEDURES

Bloaters (240-280 mm total length) and lake trout (500-700 mm) were collected by gill net each fall (September-October) in southeastern Lake Michigan, off Saugatuck, Michigan. Coho salmon (600-750 mm) were collected in east-central Lake Michigan near Ludington, Michigan, in 1972, near the entry to Portage Lake in 1973, and from the lower weir on the Platte River in 1974.

All fish were placed in plastic bags previously determined to be free of pesticides and PCB's. The packaged fish were frozen whole or packed in ice, transported to the Great Lakes Fishery Laboratory, and stored at approximately -30° C. Before analysis, the bloaters were measured and weighed, sorted according to sex and length, grouped into 10-fish composite samples, and homogenized (whole fish) by repeatedly passing them through a meat grinder. A portion of the sample was then further homogenized in a food blender. Individual lake trout and coho salmon were measured, weighed, sexed, and the whole fish similarly homogenized.

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ANALYTICAL PROCEDURES

Ten grams of homogenized fish were mixed with anhydrous sodium sulfate and extracted by the method of Hesselberg and Johnson (ref. 5). Lipids were removed from the extract by gel permeation chromatography in an automated system similar to that described by Stalling et al. (ref. 6), after which the pesticides and PCB's were separated on either silicic acid without Celite 545 (ref. 7) or on silica gel (ref. 8). The PCB's were quantitated on a gas chromatograph equipped with electron-capture detectors (^{63}Ni, 8μc) and a 5-ft x 1/8-in. ID glass column packed with 1.5 percent OV-101 on 80-100 mesh glass beads or Chromosorb W. Recoveries of PCB's from fortified samples of fish tissue average 88 percent by the described method. We performed confirmatory analyses on each sample using a second, 10-g portion of tissue saponified in alcoholic potassium hydroxide (ref. 9).

Results were calculated by summing the heights of all peaks on the chromatogram attributable to PCB's, and comparing them with the sum of peak heights obtained from analytical standards composed of Aroclors 1248, 1254, and 1260 (1:1:1). No corrections were made for losses incurred during analysis or the presence of PCB's in blanks. All results are reported as total PCB's in wet weight of tissue.

TRENDS OF PCB's

Mean concentrations of total PCB's during 1972-74 ranged from 5.24 to 5.66 ppm in bloaters, 10.4 to 12.2 ppm in coho salmon, and 12.9 to 22.9 ppm in lake trout (table 1). A plot of mean PCB concentrations in bloaters versus year of collection (figure 1) suggests no change in PCB's during the 3 years of sampling. Statistical analysis involving Student's t test (ref. 10) and the annual samples representing 110 to 160 fish showed that minor differences between mean PCB concentrations for the three sample dates were not significant. We therefore conclude that mean PCB concentrations have remained essentially unchanged since 1972 in large, adult bloaters off Saugatuck, Michigan.

A plot of mean PCB concentrations in coho salmon from 1972 to 1974 also fails to show any definite change pattern (figure 2). The mean concentrations of PCB's in these fish appear to have varied randomly between 10.4 and 12.2 ppm. Statistical analyses show that the decline between 1973 and 1974 is highly significant ($P > 0.01$) but that the 1972 samples do not differ significantly from either the 1973 or 1974 samples. Thus despite the evidence of significant variation among the three sample groups, there is no statistical evidence of a

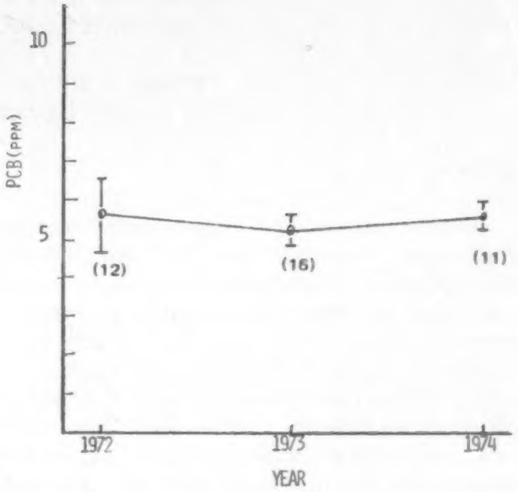

Figure 1. Trends of PCB's in Lake Michigan bloaters off Saugatuck, Michigan. Mean concentrations in whole fish (wet weight) with 95 percent confidence intervals (vertical lines). Number of 10-fish composites shown in parentheses.

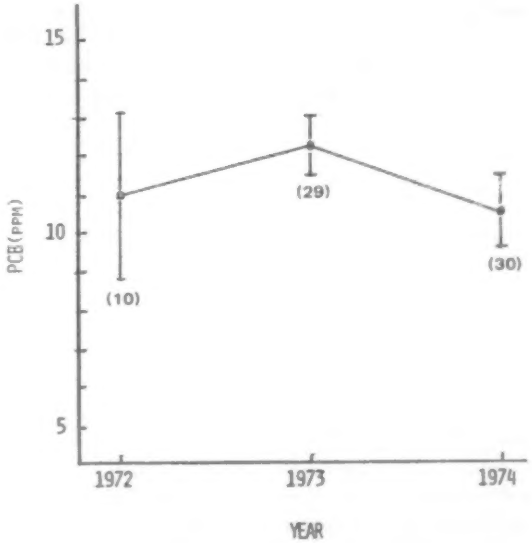

Figure 2. Trends of PCB's in coho salmon from eastern Lake Michigan. Mean concentrations in whole fish (wet weight) with 95 percent confidence in intervals (vertical lines). Number of fish in parentheses.

Table 1. Concentrations of PCB's in fall collections of Lake Michigan bloaters and lake trout taken off Saugatuck, Michigan, and coho salmon from between Ludington, Michigan, and the Platte River

Species and Year	Date collected	Number of fish	Mean length (mm)	Mean weight (g)	Mean lipid (percent)	Range of total PCB's (ppm)	Mean total PCB's (ppm)[a]
Bloater							
1972	10/19	120(12)[b]	255	177	21.6	3.12-8.17	5.66(0.95)
1973	9/27	160(16)[b]	250	165	20.0	4.20-6.33	5.24(0.37)
1974	10/9	110(11)[b]	257	175	21.6	4.83-6.22	5.57(0.31)
Coho salmon							
1972	9/19	10	693	4,156	7.3	4.93-15.4	10.9(2.1)
1973	9/21	29	620	2,967	6.0	8.24-16.6	12.2(0.8)
1974	10/1	30	665	3,148	5.4	6.99-16.3	10.4(0.9)
Lake trout							
1972	9/28	9	648	2,576	18.5	3.53-25.3	12.9(4.8)
1973	9/19	30	602	2,353	16.0	9.36-30.6	18.9(2.1)
1974	10/9	30	616	2,514	16.5	7.05-47.4	22.9(3.7)

[a] Concentrations in whole fish, wet weight, with 95 percent confidence interval in parentheses.

[b] Number of composite samples, 10 fish/sample, given in parentheses.

general increase or decrease during 1972-74 in mean PCB concentrations in coho salmon from eastern Lake Michigan.

Mean concentrations of PCB's in lake trout collected off Saugatuck, Michigan, appear to have increased from 1972 to 1974 (figure 3). Although statistical analyses show no significant differences between consecutive years, the increase between 1972 and 1974 is highly significant (P >0.005). Because of the comparatively small number of lake trout collected in 1972 (9 fish) and the high variability in PCB residues generally encountered between individual lake trout (table 1), the level of confidence in the results is low and interpretation of the apparent trend must be approached with considerable caution. Although PCB's appear to have increased in lake trout, there is no evidence of an upward trend of residues in bloaters (which represent the most reliable indicator of trends) or coho salmon; consequently additional sampling will be required to determine if the apparent upward trend of PCB's in lake trout is real. We can safely conclude, however, that mean PCB concentrations have not declined in lake trout taken off Saugatuck, Michigan.

TRENDS OF OTHER CHLORINATED HYDROCARBONS

Mean concentrations of DDT and its metabolites (total DDT) in the same samples of Lake Michigan fishes employed for the PCB study declined 69, 48, and 26 percent respectively in bloaters, coho salmon, and in lake trout during the period 1972-74 (ref. 11). Total DDT residues in fish decreased significantly within 1 year after the ban (1969-70) on the use of DDT in the

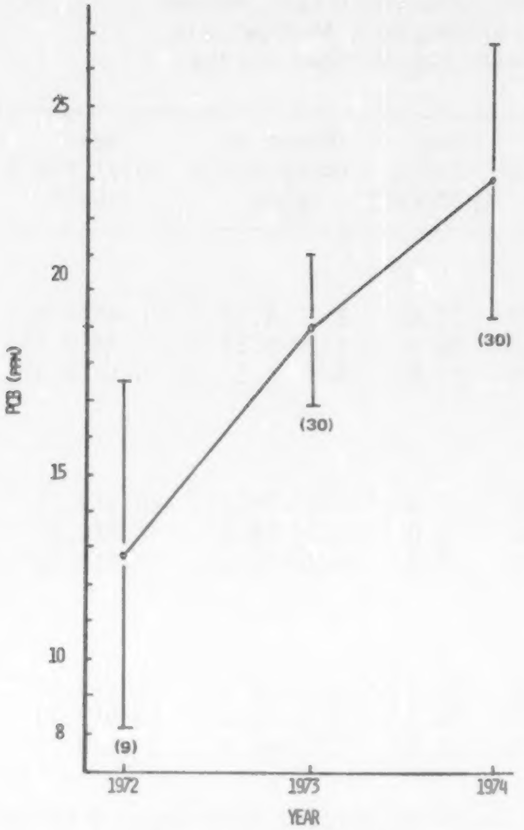

Figure 3. Trends of PCB's in Lake Michigan Lake trout off Saugatuck, Michigan. Mean concentrations in whole fish (wet weight) with 95 percent confidence intervals (vertical lines). Number of fish in parentheses.

States bordering Lake Michigan. During 1970-74, mean concentrations of total DDT dropped 87, 73, and 56 percent in bloaters, coho salmon, and lake trout respectively.

Mean concentrations of dieldrin in the same fish samples showed no definite trend from 1970 to 74 (ref. 11). The lack of change is probably due to the common use of dieldrin during the early 1970's. The rapid reduction of DDT residues in Lake Michigan fishes and the lower bioaccumulation characteristics of dieldrin lead us to believe that the recent ban (1974) on the manufacture and use of aldrin and dieldrin will result in a rapid decline of dieldrin residues in Lake Michigan fishes.

SUMMARY AND CONCLUSIONS

Mean concentrations of PCB's in Lake Michigan bloaters collected off Saugatuck, Michigan, have remained essentially unchanged since yearly analyses were initiated in 1972. Similarly, PCB residues in coho salmon from eastern Lake Michigan show no definite pattern of change. Residues of PCB's in lake trout collected off Saugatuck, Michigan, are highly variable and indicate a possible, but unconfirmed, upward trend in mean PCB concentrations. Thus, voluntary restrictions in 1970-71 on the sale of PCB's ithe United States have not led to a decrease in PCB concentrations in the Lake Michigan fishes sampled.

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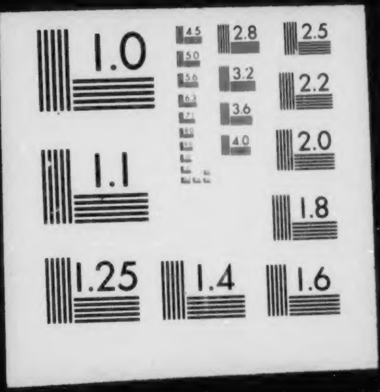

A NOTE ON POLYCHLORINATED BIPHENYLS IN AIR

Frederick W. Kutz, Ph.D.* and Henry S. C. Yang

Abstract

Samples of ambient air were collected using an ethylene-glycol impinger sampler, and analyzed for selected pesticides and polychlorinated biphenyls in suburban locations in Florida, Mississippi, and Colorado. Preliminary results for samples taken in April, May, and June of 1975 show that PCB's were present at all locations.

Samples of ambient air were collected and analyzed for selected pesticides and polychlorinated biphenyls. Suburban locations in Miami, Florida, Jackson, Mississippi, and Fort Collins, Colorado, were sampled using an ethylene-glycol impinger sampler essentially the same as reported by Enos et al. (ref. 1) except that an improvement of air flow rate was made. The air flow rate was maximized and calibrated at ca. 30 l/min. Each sampler was operated for a 24-hour period to collect one sample. The analyses of samples were done by extraction with hexane followed by cleanup with fluorsil column chromatography, using a mixture of diethyl ether and petroleum ether for elution; the fraction containing the polychlorinated biphenyls and some organochlorine pesticides underwent further separation by silicic acid column chromatography. Quantitation of the polychlorinated biphenyls was done by summation of major peaks employing the dual-column gas chromatography with electron capture detector.

Preliminary results for samples taken in April, May, and June of 1975 show that PCB's were present at all locations. Although the data varied, the average concentration at each of the three locations was approximately 100 nanograms per cubic meter. The presence of polychlorinated biphenyls in ambient air samples taken in Miami and Jackson has been confirmed by combined gas chromatography-mass spectrometry. Initial identification of the polychlorinated biphenyls indicates that they were most comparable to the Aroclor 1254 standard.

In July the number of sampling sites was expanded to five with the addition of Lafayette, Indiana, and Harrisburg, Pennsylvania. Results from all sites should be available in the near future.

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POLYCHLORINATED BIPHENYLS IN THE SURFACE WATERS AND BOTTOM SEDIMENTS OF THE MAJOR DRAINAGE BASINS OF THE UNITED STATES

D. Steve Dennis, Ph.D*

Abstract

Data gathered from monitoring activities indicate the widespread occurrence of PCB's in surface waters and bottom sediments of the major drainage basins of the United States. A preliminary assessment of PCB levels shows median residue levels of the positive detections for the years 1971 to 1974 ranging between 0.1 to 3.0 µg/l for unfiltered water samples and from 1.2 to 160.0 µg/kg for bottom sediments. The highest levels were found in basins east of the Mississippi and bottom sediments may contain concentrations of PCB's many times higher than those in the overlying water.

INTRODUCTION

Within the past few years, particularly in the past year, polychlorinated biphenyls (PCB's) have been identified as a major contaminant of natural waters. PCB's in the hydrologic environment can originate from several sources including: (1) runoff from sewage sludges disposed of on land, (2) discharge of industrial and municipal wastes (this includes treated as well as untreated wastes), (3) accidental spills or improper waste disposal practices, and (4) formerly as ingredients of pesticides or as carriers for pesticides. Probably the largest amount of PCB's in the environment results from industrial and municipal discharges to inland and coastal waters (refs. 1–5).

Although much attention has been focused on estimating levels of PCB's in aquatic organisms, and the potential hazards involved, few data are available on the occurrence of PCB's in water and bottom sediments. The U.S. Department of Interior's Geological Survey and the U.S. Environmental Protection Agency's (EPA) Office of Pesticide Programs, Technical Services Division, through their National Water Monitoring Program for Pesticide Residues and various water-resource-assessment projects, are accumulating data which permit a preliminary assessment of PCB contamination of the Nation's drainage basins. The data presented here were gathered by the Geological Survey. They show the occurrence of PCB's (PCB mixtures predominately resemble Aroclor 1254) in whole water samples (i.e., water samples which are unfiltered) and in bottom sediments in the 17 major drainage basins of the United States and Puerto Rico (figure 1) for the years 1971 to 1974. All data were obtained from STORET, which is EPA's computerized information system that draws on both State and Federal agencies for parametric data relating to water quality.

Although there is not a nationwide program specifically designed to assess PCB levels in the aquatic environment, the present Geological Survey and EPA water monitoring programs are expanding and gradually increasing to more adequately determine PCB levels in the surface waters of the United States. These programs are also being designed to determine both national and regional trends in the concentration of PCB's.

METHODS AND PROCEDURES

Water samples were collected either by depth integration at the center of flow or by the equal-transit-rate method. In the latter method a standard suspended-sediment sampler, such as the US D-49 suspended-sediment sampler, was used to collect a velocity- and discharge-weighted sample. Samples were taken at a number of equally spaced verticals across a stream, depending on stream width (usually three sites were sampled across a stream). The transit rate of movement of the sampler from the water surface to the stream bed and back to the surface was the same at all verticals. Samples collected at each vertical were composited into a single sample intended to represent the entire flow across the stream.

Bottom sediments were sampled by collecting a series of cores across the stream bed using piston type or US BMH-60 or US BM-54 bed material samplers or a commercial core sampler. When a core sampler was used, cores were composited to form a representative sample of the stream bed (see refs. 5,6 for a description of sampling procedures).

Once the samples were collected, PCB residues were analyzed by the multiple-pesticide-residue methods for water, suspended sediment, and bottom sediment described by Goerlitz and Brown (ref. 7).

Basic identification of PCB residues was made by dual-column electron capture/gas-liquid chromatography and confirmed by gas-liquid chromatography or mass spectrometry. The amount of PCB's was determined by matching the peaks on the chromatogram with the nearest commercial formulation. The reported values are subject to some error because of the complexity of multiple peaks resulting from mixtures of PCB's in environmental samples.

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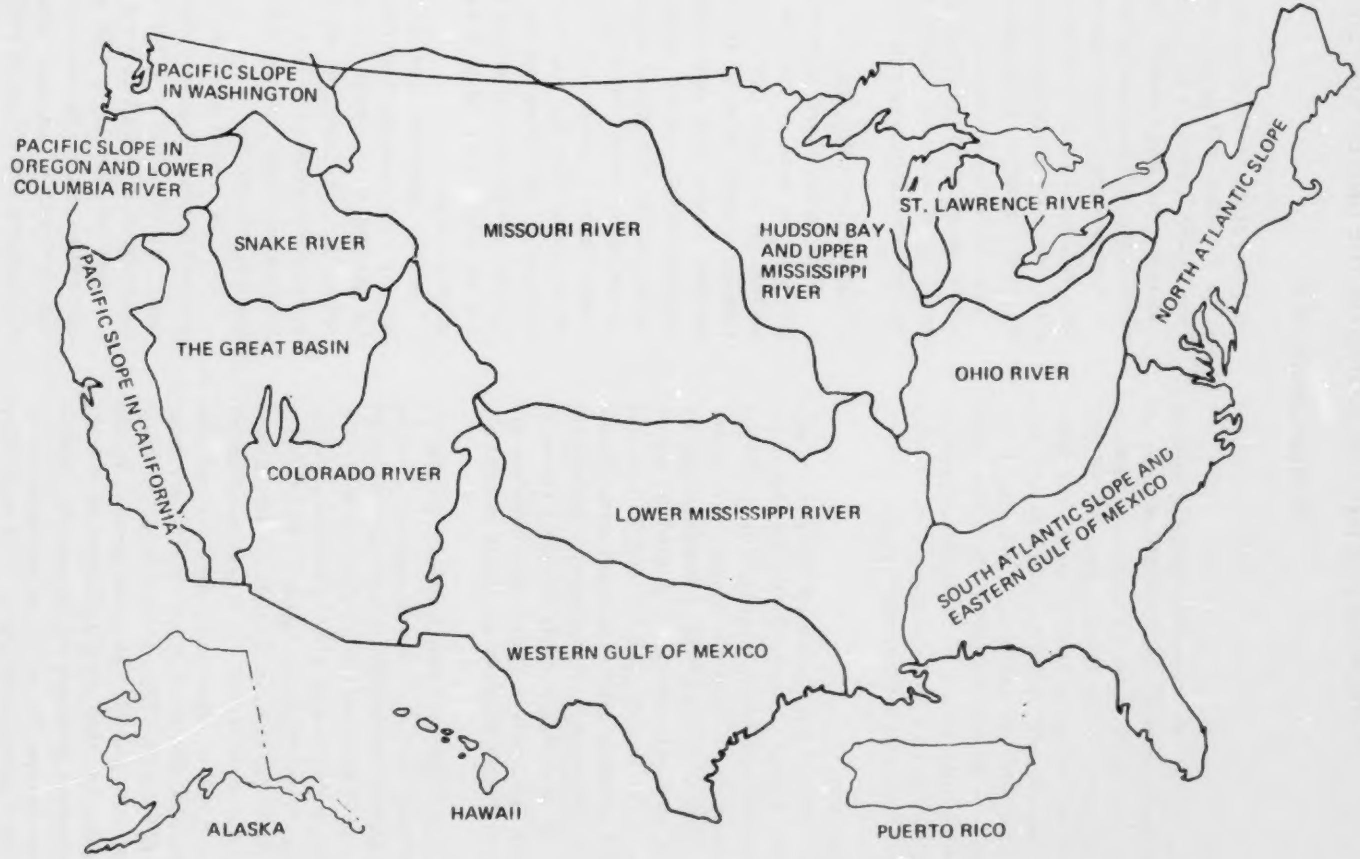
PACIFIC SLOPE IN WASHINGTON
PACIFIC SLOPE IN OREGON AND LOWER COLUMBIA RIVER
SNAKE RIVER
THE GREAT BASIN
PACIFIC SLOPE IN CALIFORNIA
COLORADO RIVER
MISSOURI RIVER
HUDSON BAY AND UPPER MISSISSIPPI RIVER
ST. LAWRENCE RIVER
NORTH ATLANTIC SLOPE
OHIO RIVER
LOWER MISSISSIPPI RIVER
SOUTH ATLANTIC SLOPE AND EASTERN GULF OF MEXICO
WESTERN GULF OF MEXICO
ALASKA
HAWAII
PUERTO RICO

Figure 1. Map showing the major drainage basins in the United States and Puerto Rico.

Table 1. PCB's in whole water samples in the major drainage basins of the United States and Puerto Rico for 1971

Drainage basin	Sample size	% of positive detections	Median of positive detections in μg/l	Range of positive detections in μg/l
1. North Atlantic Slope	12	58.3	0.2	0.1 - 2.1
2. South Atlantic Slope and Eastern Gulf of Mexico	12	0	ND	ND
3. Ohio River	-	-	-	-
4. St. Lawrence River	15	26.7	0.3	0.1 - 0.5
5. Hudson Bay and Upper Mississippi River	-	-	-	-
6. Missouri River	1	0	ND	ND
7. Lower Mississippi	1	100.0	3.0	3.0
8. Western Gulf of Mexico	9	44.4	0.3	0.1 - 0.7
9. Colorado River	-	-	-	-
10. Great Basin	-	-	-	-
11. Pacific Slope Basins in California	-	-	-	-
12. Pacific Slope Basins in Washington	-	-	-	-
13. Snake River	-	-	-	-
14. Pacific Slope Basins in Oregon and Lower Columbia River	-	-	-	-
15. Alaska	-	-	-	-
16. Hawaii	-	-	-	-
17. Puerto Rico	-	-	-	-

Note: - = no samples; ND = not detected.

Table 2. PCB's in whole water samples in the major drainage basins of the United States and Puerto Rico for 1972

Drainage basin	Sample size	% of positive detections	Median of positive detections in μg/l	Range of positive detections in μg/l
1. North Atlantic Slope	112	17.0	0.1	0.1 - 0.3
2. South Atlantic Slope and Eastern Gulf of Mexico	122	0.8	ND	ND
3. Ohio River	10	20.0	0.2	0.1 - 0.2
4. St. Lawrence River	29	13.8	0.1	0.1 - 0.2
5. Hudson Bay and Upper Mississippi River	12	16.7	0.3	0.2 - 0.3
6. Missouri River	34	0	ND	ND
7. Lower Mississippi	39	2.6	0.1	0.1
8. Western Gulf of Mexico	93	8.6	0.3	0.1 - 2.6
9. Colorado River	24	0	ND	ND
10. Great Basin	9	0	ND	ND
11. Pacific Slope Basins in California	20	0	ND	ND
12. Pacific Slope Basins in Washington	14	0	ND	ND
13. Snake River	-	-	-	-
14. Pacific Slope Basins in Oregon and Lower Columbia River	3	0	ND	ND
15. Alaska	-	-	-	-
16. Hawaii	1	0	ND	ND
17. Puerto Rico	3	0	ND	ND

Note: - = no samples; ND = not detected.

Table 3. PCB's in whole water samples in the major drainage basins of the United States and Puerto Rico for 1973

Drainage basin	Sample size	% of positive detections	Median of positive detections in µg/l	Range of positive detections in µg/l
1. North Atlantic Slope	109	3.7	0.1	0.1
2. South Atlantic Slope and Eastern Gulf of Mexico	114	4.4	0.1	0.1 - 20.0
3. Ohio River	6	0	ND	ND
4. St. Lawrence River	31	3.2	0.1	0.1
5. Hudson Bay and Upper Mississippi River	30	10.0	0.2	0.1 - 0.4
6. Missouri River	95	0	ND	ND
7. Lower Mississippi	291	0.7	1.6	0.2 - 2.9
8. Western Gulf of Mexico	362	1.7	0.3	0.1 - 0.6
9. Colorado River	64	0	ND	ND
10. Great Basin	12	0	ND	ND
11. Pacific Slope Basins in California	73	0	ND	ND
12. Pacific Slope Basins in Washington	33	0	ND	ND
13. Snake River	2	0	ND	ND
14. Pacific Slope Basins in Oregon and Lower Columbia River	9	0	ND	ND
15. Alaska	-	-	-	-
16. Hawaii	1	0	ND	ND
17. Puerto Rico	37	0	ND	ND

Note: - = no samples; ND = not detected.

Table 4. PCB's in whole water samples in the major drainage basins of the United States and Puerto Rico for 1974

Drainage basin	Sample size	% of positive detections	Median of positive detections in µg/l	Range of positive detections in µg/l
1. North Atlantic Slope	51	2.0	0.1	0.1
2. South Atlantic Slope and Eastern Gulf of Mexico	151	0	ND	ND
3. Ohio River	24	0	ND	ND
4. St. Lawrence River	38	0	ND	ND
5. Hudson Bay and Upper Mississippi River	33	0	ND	ND
6. Missouri River	28	0	ND	ND
7. Lower Mississippi	269	2.6	0.1	0.1 - 0.2
8. Western Gulf of Mexico	325	1.2	0.3	0.2 - 0.7
9. Colorado River	52	0	ND	ND
10. Great Basin	12	0	ND	ND
11. Pacific Slope Basins in California	74	2.7	0.1	0.1
12. Pacific Slope Basins in Washington	13	7.7	0.1	0.1
13. Snake River	11	0	ND	ND
14. Pacific Slope Basins in Oregon and Lower Columbia River	8	0	ND	ND
15. Alaska	-	-	-	-
16. Hawaii	1	0	ND	ND
17. Puerto Rico	68	1.5	0.8	0.8

Note: - = no samples; ND = not detected.

Table 5. PCB's in bottom sediments in the major drainage basin of the United States and Puerto Rico for 1971

Drainage basin	Sample size	% of positive detections	Median of positive detections in µg/kg	Range of positive detections in µg/kg
1. North Atlantic Slope	3	100.0	50.0	10.0 - 100.0
2. South Atlantic Slope and Eastern Gulf of Mexico	19	63.2	64.5	10.0 - 200.0
3. Ohio River	1	100.0	8.8	8.8
4. St. Lawrence River	1	100.0	10.0	10.0
5. Hudson Bay and Upper Mississippi River	-	-	-	-
6. Missouri River	2	100.0	1.2	0.3 - 2.0
7. Lower Mississippi	4	100.0	30.0	10.0 - 80.0
8. Western Gulf of Mexico	20	100.0	10.0	2.0 - 290.0
9. Colorado River	-	-	-	-
10. Great Basin	-	-	-	-
11. Pacific Slope Basins in California	-	-	-	-
12. Pacific Slope Basins in Washington	-	-	-	-
13. Snake River	-	-	-	-
14. Pacific Slope Basins in Oregon and Lower Columbia River	-	-	-	-
15. Alaska	-	-	-	-
16. Hawaii	-	-	-	-
17. Puerto Rico	-	-	-	-

Note: - = no samples; ND = not detected.

Table 6. PCB's in bottom sediments in the major drainage basins of the United States and Puerto Rico for 1972

Drainage basin	Sample size	% of positive detections	Median of positive detections in μg/kg	Range of positive detections in μg/kg
1. North Atlantic Slope	56	57.1	10.0	5.0 - 800.00
2. South Atlantic Slope and Eastern Gulf of Mexico	101	32.7	30.0	5.0 - 500.00
3. Ohio River	-	-	-	-
4. St. Lawrence River	10	90.0	20.0	2.0 - 800.0
5. Hudson Bay and Upper Mississippi River	3	0	ND	ND
6. Missouri River	22	81.8	2.0	2.0
7. Lower Mississippi	25	48.0	2.0	2.0 - 2400.0
8. Western Gulf of Mexico	66	43.9	4.0	2.0 - 250.0
9. Colorado River	7	71.4	2.0	2.0
10. Great Basin	9	66.7	2.0	2.0
11. Pacific Slope Basins in California	6	83.3	20.0	2.0 - 190.0
12. Pacific Slope Basins in Washington	9	44.4	2.0	2.0
13. Snake River	1	0	ND	ND
14. Pacific Slope Basins in Oregon and Lower Columbia River	1	100.0	2.0	2.0
15. Alaska	-	-	-	-
16. Hawaii	1	0	ND	ND
17. Puerto Rico	-	-	-	-

Note: - = no samples; ND = not detected.

Table 7. PCB's in bottom sediments in the major drainage basins of the United States and Puerto Rico for 1973

Drainage basin	Sample size	% of positive detections	Median of positive detections in µg/kg	Range of positive detections in µg/kg
1. North Atlantic Slope	58	70.7	10.0	4.0 - 4000.0
2. South Atlantic Slope and Eastern Gulf of Mexico	123	48.8	10.0	5.0 - 600.0
3. Ohio River	4	25.0	20.0	20.0
4. St. Lawrence River	24	91.7	20.0	5.0 -13000.0
5. Hudson Bay and Upper Mississippi River	14	0	ND	ND
6. Missouri River	17	0	ND	ND
7. Lower Mississippi	57	1.8	20.0	20.0
8. Western Gulf of Mexico	188	3.7	50.0	14.0 - 180.0
9. Colorado River	4	0	ND	ND
10. Great Basin	7	0	ND	ND
11. Pacific Slope Basins in California	14	0	ND	ND
12. Pacific Slope Basins in Washington	12	0	ND	ND
13. Snake River	1	0	ND	ND
14. Pacific Slope Basins in Oregon and Lower Columbia River	-	-	-	-
15. Alaska	2	0	ND	ND
16. Hawaii	1	0	ND	ND
17. Puerto Rico	6	16.7	5.0	5.0

Note: - = no samples; ND = not detected.

Table 8. PCB's in bottom sediments in the major drainage basins of the United States and Puerto Rico for 1974

Drainage basins	Sample size	% of positive detections	Median of positive detections in µg/kg	Range of positive detections in µg/kg
1. North Atlantic Slope	99	56.6	31.0	2.0 - 800.0
2. South Atlantic Slope and Eastern Gulf of Mexico	171	25.7	11.0	2.0 - 530.0
3. Ohio River	6	33.3	35.0	9.0 - 61.0
4. St. Lawrence River	40	60.0	20.0	3.0 - 700.0
5. Hudson Bay and Upper Mississippi River	26	0	ND	ND
6. Missouri River	5	0	ND	ND
7. Lower Mississippi	81	9.9	10.5	1.0 - 39.0
8. Western Gulf of Mexico	157	21.7	27.0	2.0 - 330.0
9. Colorado River	3	0	ND	ND
10. Great Basin	4	0	ND	ND
11. Pacific Slope Basins in California	26	11.5	2.0	2.0 - 65.0
12. Pacific Slope Basins in Washington	5	0	ND	ND
13. Snake River	4	0	ND	ND
14. Pacific Slope Basins in Oregon and Lower Columbia River	1	0	ND	ND
15. Alaska	-	-	-	-
16. Hawaii	-	-	-	-
17. Puerto Rico	18	27.2	160.0	10.0 - 640.0

Note: - = no samples; ND = not detected.

RESULTS

Tables 1 through 8 present the PCB residue data by year for water and bottom sediments. For each year the following is given: number of samples, percent of positive detections, median of positive detections, and maximum of positive detections in µg/l for water and µg/kg for bottom sediments (one µg/l and one µg/kg are roughly equivalent to one ppb).

Not all drainage basins had detectable levels of PCB's in both water and bottom sediments. However, it should be noted that this may be in part due to the location of sampling stations within drainage basins and/or the small number of samples within drainage basins.

Median residue levels of the positive detections in whole water samples ranged from 0.2 to 3.0 µg/l, 0.1 to 0.3 µg/l, 0.1 to 1.6 µg/l, and 0.1 to 0.8 for all drainage basins for 1971, 1972, 1973, and 1974, respectively (tables 1 to 4). For both these data and the data for each basin there is no indication of an increase in PCB levels and in many instances the levels decreased. The highest PCB level was found in the South Atlantic Slope and Eastern Gulf of Mexico basin and in general, the highest levels were found in basins east of the Mississippi. One might speculate that this is due to the presence of certain industries in the eastern United States.

Bottom sediments showed a pattern similar to that of water with the highest PCB residue levels occurring in the eastern United States (table 5 to 8). Median residue levels of the positive detections for all basins ranged from 1.2 to 64.5 µg/kg, 2.0 to 30.0 µ/kg, 5.0 to 50.0 µg/kg, and 2.0 to 160.0 µg/kg for 1971, 1972, 1973, and 1974, respectively. The data indicate that, as with the whole water samples, the levels decreased in some basins. However, it appears as if PCB's are more persistent in bottom sediments.

DISCUSSION

The data which I have presented here indicate that PCB's are widespread in the surface waters and bottom sediments of the Nation. Crump-Wiesner et al. (ref. 9) also found PCB's to have a ubiquitous occurrence and distribution in surface water, ground water, and bottom sediments. According to Veith and Lee (ref. 8) ". . . the chlorinated biphenyls may be one of the more widespread. . ." environmental contaminants.

It is well documented that bottom sediments may contain concentrations of pesticide residues many times higher than the overlying water (refs. 10-13). The data presented here and previously published data (refs. 1,4,9,14) indicate that a similar relationship holds true for PCB's. Even in drainage basins, where PCB's were not detected in water samples such as the Missouri River, they were present in bottom sediments. It is assumed that this is due to the low water solubility and high specific gravity of PCB's (ref. 14). According to Nisbet and Sarofim (ref. 14), ". . . it is expected that most of the PCBs discharged into the environment will be resting as sludges or adsorbed in the sediment at the bottom of rivers or lakes near their point of discharge, and that transport in streams will be primarily by means of waterborne particles."

Finally, it is important to note that although PCB's are found in water and bottom sediments, this information must be related to the entire aquatic environment before the data can be adequately evaluated. This is because (1) PCB's in solution and sorbed on organic and inorganic particles are available for introduction into food chains, (2) PCB's are accumulated in the tissues of animals which are exposed to water and bottom sediments containing PCB's, (3) biological magnification may occur in food chains, and (4) all components in an aquatic ecosystem are interrelated.

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PCB's IN AGRICULTURAL AND URBAN SOIL

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Abstract

Polychlorinated biphenyls in soil have been monitored since 1972 as part of the National Soils Monitoring Program, originally established to measure pesticide residue levels in agricultural soils, raw agricultural commodities, and urban soils across the Nation. The PCB's are monitored as part of this program because of their chemical similarity to certain chlorinated pesticides.

The PCB's have rarely been detected in agricultural soils of the United States. Only 0.1 percent of the soil samples collected in the National Soils Monitoring Program for 1972 contained detectable PCB levels. However, detectable levels of PCB's occur more frequently in urban soils. Of the 19 metropolitan areas sampled since 1971, 12 of the cities, or 63 percent showed detectable PCB levels. The most commonly encountered PCB was Aroclor 1254, which was identified in approximately 40 percent of the positive samples, while Aroclor 1260 was prevalent in about 20 percent of the positive samples. In the remaining 40 percent of the positive samples, no identification was made of the specific Aroclor. The occurrence of these compounds appears to be more prevalent in the "urban" portion (within the city limits) than in the "suburban" portion (remainder) of these metropolitan areas.

INTRODUCTION

The occurrence of polychlorinated biphenyls (PCB's) in environmental media has been known and well-documented for several years; it is only recently that the magnitude of the contamination has created widespread concern.

These compounds have been monitored in agricultural and urban soils since 1972 as part of the National Soils Monitoring Program. This program was established to measure pesticide residue levels in agricultural soils, raw agricultural commodities, and urban soils across the Nation. It was initiated by the U.S. Department of Agriculture and is now operated by the U.S. Environmental Protection Agency. The PCB's are monitored as part of this program because of their chemical similarity to certain chlorinated pesticides.

SAMPLING PROCEDURES

For cropland sampling, 50 cores 5.1 x 7.6 cm in size were collected over an evenly spaced 5 x 10 grid on each 4-hectare site (ref. 1). In urban sampling, this procedure was modified to collect 16 cores on a 4 x 4 grid over a 225-m^2 plot (15 x 15 m) (ref. 2). Field processing was similar for both types of samples; the cores were composited, thoroughly mixed, and sieved through a 6.3-mm mesh, and a 2-l subsample was sent to the EPA Technical Services Division's Pesticide Monitoring Laboratory in Bay St. Louis, Mississippi, for chemical analysis.

ANALYTICAL PROCEDURES

Sample Preparation

A 300-g subsample was dampened with water and extracted with 600 ml of 3:1 hexane:isopropanol solvent. The isopropanol was removed by three distilled water washes and the hexane extract was dried through anhydrous sodium sulfate and stored at low temperature for subsequent gas-liquid chromatographic analysis.

Gas-Liquid Chromatography

Analyses were performed on gas chromatographs equipped with tritium foil electron affinity detectors. A multiple system of polar and nonpolar columns was utilized for identification and confirmation. Instrument parameters were as follows:

Columns:

Glass, 6 mm o.d. x 4 mm i.d., 183-cm long, packed with one of the following: 9% QF-1 on 100/120-mesh Gas-Chrom Q; 3% DC-200 on 100/120-mesh Gas-Chrom Q; or 1.5% OV-17/1.95% QF-1 on 100/120-mesh Sepulcoport.

Carrier Gases:

5% methane-argon at a flow rate of 80 ml/min; prepurified nitrogen at a flow rate of 80 ml/min.

Temperatures:

Detector	200°C
Injection port	250°C
Column QF-1	166°C
Column DC-200	170°–175°C
Mixed column	185°–190°C.

Sensitivity or minimum detection levels were 0.05 to 0.1 ppm. The average recovery rate in soil was 90 to 110 percent.

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DISCUSSION

Agricultural Soils

PCB's have rarely been detected in the Nation's agricultural soils. Of the 1,556 soil samples collected in 1972 as part of the National Soils Monitoring Program, only 2 samples or 0.1 percent contained detectable levels of PCB's.

Urban Soils

Detectable levels of PCB's occur more frequently in urban soils than in agricultural soils. Of the 19 metropolitan areas sampled since 1971, 12 of the cities, or 63 percent showed detectable levels of PCB's. Corresponding data are presented in tables 1-4. The most commonly encountered PCB was Aroclor 1254, which was identified in 39 percent of the positive samples, while Aroclor 1260 was prevalent in 21 percent of the positive samples. Although no attempt was made to identify the specific Aroclor in 39 percent of the positive samples, it is likely that a majority contained Aroclor 1254.

All sites sampled within a metropolitan area were classified into two of four categories. First, sites were identified as either "lawn" or "waste," to indicate whether or not they seemed likely to receive regular maintenance. Secondly, each site was classified as urban if it was within the city limits, and suburban if it was located in the remainder of the metropolitan area.

When the positive sites in the 12 cities were categorized in this manner, 42 percent were lawn sites (maintained), while 58 percent were waste sites (not maintained). This difference was not considered significant. However, the second categorization showed that 70 percent of the PCB-positive sites were urban, or within the city limits, whereas only 30 percent were classified as suburban sites. In comparison, the ratio of urban to suburban sites for these 19 cities was 37 percent to 63 percent.

In summary, PCB occurrence in agricultural soils is extremely rare and is not considered a problem. However, the occurrence of these compounds is more prevalent in metropolitan areas, particularly within city limits, and monitoring should be continued to determine trends over time.

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Table 1. Occurrence of polychlorinated biphenyls in soils of five U.S. cities sampled in 1971. U.S. Environmental Protection Agency Urban Monitoring Program

Location	Total No. of sites	No. of positive detections	Percent of positive detections	Detected values (ppm)		Arithmetic mean (ppm)	Geometric mean estimate (ppm)	PCB type-if identified
				Minimum	Maximum			
Baltimore, Md.	156	6	3.9	0.09	0.74	0.02	0.001	1260
Gadsden, Ala.	55	1	1.8	11.94		0.21	*[b]	not identified
Hartford, Conn.	48	ND[a]	-					
Macon, Ga.	43	ND	-					
Newport News, Va.	78	1	1.3	3.30		0.04	*	1254

[a] ND = Not detected.

[b] * = Geometric mean estimate not calculated when less than two positive values present.

Table 2. Occurrence of polychlorinated biphenyls in soils of five U.S. cities sampled in 1972. U.S. Environmental Protection Agency Urban Monitoring Program

Location	Total No. of sites	No. of positive detections	Percent of positive detections	Detected values (ppm) Minimum	Detected values (ppm) Maximum	Arithmetic mean (ppm)	Geometric mean estimate (ppm)	PCB type-if identified
Des Moines, Iowa	82	3	3.7	0.34	0.94	0.03	0.002	not identified
Fitchburg, Mass.	35	ND[a]	-					
Lake Charles, La.	70	1	1.4	1.31		0.02	*[b]	1254
Pittsburgh, Pa.	189	1	0.5	1.01		0.01	*	not identified
Reading, Pa.	49	ND	-					

[a] ND = Not detected.

[b] * = Geometric mean estimate not calculated when less than two positive values present.

Table 3. Occurrence of polychlorinated biphenyls in soils of five U.S. cities sampled in 1973. U.S. Environmental Protection Agency Urban Monitoring Program

Location	Total No. of sites	No. of positive detections	Percent of positive detections	Detected values (ppm) Minimum	Detected values (ppm) Maximum	Arithmetic mean (ppm)	Geometric mean estimate (ppm)	PCB type-if identified
Evansville, Ind.	82	ND[a]	-					
Greenville, S.C.	86	3	3.5	0.13	1.59	0.02	0.001	1254
Pittsfield, Mass.	45	ND	-					
Tacoma, Wash.	95	6	6.3	0.18	0.63	0.03	0.003	1-1254 5-not identified
Washington, D.C.	116	2	1.7	0.32	0.80	<0.01	0.001	1-1254 1-not identified

[a] ND = Not detected.

Table 4. Occurrence of polychlorinated biphenyls in soils of five U.S. cities sampled in 1974. U.S. Environmental Protection Agency Urban Monitoring Program

Location	Total No. of sites	No. of positive detections	Percent of positive detections	Detected values (ppm)		Arithmetic mean (ppm)	Geometric mean estimate (ppm)	PCB type-if identified	Percent of Analyses Completed
				Minimum	Maximum				
Durham, N.C.	66	1	1.5	0.40		0.01	*[b]	1254	94
Gary, Ind.	89	5	5.6	0.50	3.33	<0.01	0.003	1-not identified 4-1254	100
San Francisco, Calif.	119	3	2.5	0.39	1.15	<0.01	0.001	1-not identified 1-1254 1-1260	71
Springfield, Ill.	72	ND[a]	-						100
Pine Bluff, Ark.	59	Analyses not completed							

[a] ND = Not detected.

[b] * = Geometric mean estimate not calculated when less than two positive values present.

MARINE INPUTS OF POLYCHLORINATED BIPHENYLS OFF SOUTHERN CALIFORNIA

David R. Young, Deirdre J. McDermott, and Theadore C. Heesen*

Abstract

Rates of polychlorinated biphenyl (PCB) transport via potential input routes to the coastal waters off southern California have been quantified. Submarine discharge of municipal wastewater was found to be the single largest source, contributing 5,400 kg of these synthetic organics in 1974. However, inputs via this route appear to be decreasing, as the corresponding estimate for 1971 exceeded 19,000 kg. Bottom sediments around the largest outfalls contain up to 10 ppm PCB. Dry aerial fallout also appears to be an important source. The estimated deposition rate of 1254 PCB onto the coastal waters during 1973-74 was 1,800 kg/yr; highest inputs were measured off the Los Angeles Basin. This region also contributed the most PCB in surface runoff, although less than 800 kg were discharged annually during 1972-73 via storm and dry-weather flow. Direct industrial discharges to San Pedro and San Diego Harbors did not appear to be a significant PCB source, totaling less than 250 kg/yr. Although antifouling paints may have been a very significant source in the past, present inputs are negligible. Also, despite high contamination levels measured in three major harbors, we found no evidence of significant PCB transport from these harbors to the adjacent coastal waters.

INTRODUCTION

Polychlorinated biphenyls (PCB's) have been used for more than 4 decades in a wide variety of industrial products (refs. 1,2). Within the last 10 years, the problem of environmental contamination by these synthetic organic compounds has become a subject of increasing concern (refs. 3,4,5). Here we report the results obtained to date on the routes by which PCB's enter one coastal marine ecosystem, the Southern California Bight, and the rates at which inputs occur.

MUNICIPAL WASTEWATERS

Every day, over 1 billion gallons (approximately 4×10^9 l) of municipal wastewaters are discharged from submarine outfalls to the Southern California Bight. Almost 95 percent of these wastewaters are released by five major treatment plants.† By sampling these five plants, we have been able to obtain a reasonably representative picture of PCB inputs to the Bight via this route. Resultant effluent concentrations and estimated annual mass emission rates for 1972-75 are summarized in table 1; details of sampling and analytical procedures are presented elsewhere (ref. 6). These data indicate that in recent years there has been a significant decrease in the amount of PCB's discharged through these outfall systems.

The municipal wastewater discharges have resulted in obvious contamination of the bottom sediments around the larger outfall systems. Figure 1 illustrates concentrations of total PCB measured in surface sediments collected around the Hyperion outfalls in Santa Monica Bay. The maximum concentration measured near the longer Hyperion outfall (the sludge line) and the JWPCP outfalls were similar, on the order of 10 ppm (mg/dry kg). However, within a few kilometers of the Hyperion sludge discharge, the surface sediment concentrations of PCB had fallen to about 0.2 ppm. Near the JWPCP outfalls, large vertical gradients were observed, with values decreasing by one to two orders of magnitude over a 30-cm depth. We estimate that the load of 1254 PCB in these Palos Verdes Shelf sediments (50 sq km) is on the order of 6 metric tons (ref. 7). Surface sediments around the Orange County outfall did not exhibit large gradients of total PCB in 1975; the median value was 0.1 ppm, with values ranging from 0.04 to 0.3 ppm. Similar concentrations were measured 6 months after discharge commenced in 1971, indicating little buildup of PCB's in these sediments since then.

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†These treatment plants are located near the following stations indicated in figure 2: Oxnard-Port Hueneme; Hyperion-Santa Monica; JWPCP-Palos Verdes Peninsula; Orange Co.-Newport Beach; Point Loma-midway between La Jolla and the U.S.-Mexico border.

Table 1. Representative PCB concentrations and mass emission rates[a] from major municipal wastewater discharges to the Southern California Bight, 1972-75

Discharger	Total Susp. Solids (mg/l)	Concentration (µg/l)	Mass Emission Rate (kg/yr)	Concentration (µg/l)	Mass Emission Rate (kg/yr)
1972					
JWPCP	290	24	11,600	ND	-
Hyperion					
Effluent	90	ND	-	0.3	140
Sludge	7,670	280	1,780	ND	-
Orange Co.	150	28	5,800	ND	-
Point Loma	140	ND	-	0.9	120
Oxnard	70	ND	-	0.2	3
Total			>19,180		>260
1973					
JWPCP	260	3.9	1,930	1.6	790
Hyperion					
Effluent	80	ND	-	0.38	180
Sludge	7,500	ND	-	26	170
Orange Co.	150	ND	-	1.4	290
Point Loma	160	ND	-	0.48	70
Oxnard	130	ND	-	0.35	6
Total			>1,930		1,510
1974					
JWPCP	280	1.9	910	0.76	360
Hyperion					
Effluent	80	0.11	50	0.24	110
Sludge	7,300	72	470	50	320
Orange Co.	110	8.0	1,890	0.89	210
Point Loma	140	6.6	950	0.74	110
Oxnard	170	0.1	1	0.24	3
Total			4,270		1,110
1975					
JWPCP		0.6	290	0.52	250
Hyperion					
Effluent		0.06	30	0.18	80
Sludge		34	220	28	180
Orange Co.		5.2	1,230	0.63	150
Point Loma		1.1	160	0.39	60
Oxnard		0.03	0.4	0.16	2
Total			1,930		720

[a] The following 1974 flow values (million gallons per day) were used for calculations of preliminary 1975 mass emission rates: JWPCP--350; Hyperion effluent--340; Hyperion sludge--4.7; Orange Co.--170; Point Loma--100; Oxnard--10.

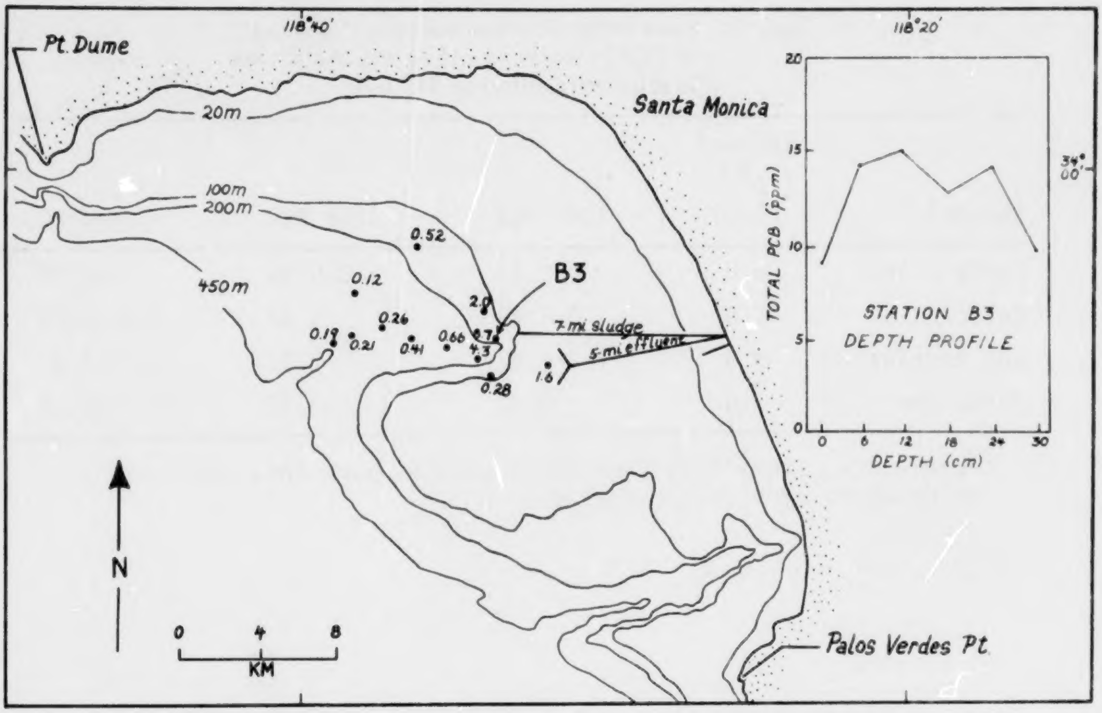

Figure 1. Concentrations of total PCB (mg/dry kg) measured in the top 2 cm of bottom sediments collected during July 1971 off the Hyperion submarine outfalls in Santa Monica Bay.

We have found that benthic organisms trawled from these regions are highly contaminated with PCB's. Crab and flatfish muscle tissue contain on the order of 1 ppm total PCB, approximately 100 times the levels measured in specimens from uncontaminated control stations. Details of these studies are presented elsewhere in these proceedings (ref. 8) and a background paper (ref. 9).

SURFACE RUNOFF

In water year 1971-72, we conducted a detailed survey of chlorinated hydrocarbon inputs to the Southern California Bight via storm runoff. During the major storms of the year, time series of depth-integrated samples were collected in an all-metal sampler near the mouths of four major drainage channels—the Santa Clara River, Ballona Creek, the Los Angeles River, and the Santa Ana River.* Analyses of the preserved samples were conducted by Dr. R. Risebrough and B. de Lappe at Bodega Marine Laboratory (University of California at Berkeley). The following year, we resurveyed one of these channels on a limited basis. Los Angeles River runoff was sampled during four storms and analyzed in our laboratory. Subsequently, two seasonal collections of dry-weather flow were made from channels located throughout the Bight (ref. 6).

Table 2 presents final flow-weighted mean PCB concentrations in 1971-72 storm runoff in the four channels samples. In table 3 we list flow-weighted mean PCB concentrations for the four Los Angeles River storm flows sampled in 1972-73, and compare the yearly averages for this channel with those for 1971-72. To aid in the comparison, we also list corresponding results for total DDT and dieldrin. In light of the variation measured in concentrations during any given storm, and the fact that two water years and two laboratories were involved, the agreement between the flow-weighted means is remarkably good. Because of this general agreement, we concluded that the data from the 1971-72 Bight-wide storm runoff survey could be extrapolated to the following year, on the basis of relative flow rates.

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*These channels discharge surface runoff near the following stations indicated in figure 2: Santa Clara R.-Port Hueneme; Ballona Ck.-Santa Monica; Los Angeles R.-midway between Palos Verdes Peninsula and Newport Beach; Santa Ana R.-Newport Beach.

Table 2. Flow-weighted mean concentrations ($\mu g/l$) of PCB in storm runoff via major channels in southern California, 1971-72

Channel	Volume[a] (10^6 cu m)	1242 PCB	1254 PCB	Total PCB
Santa Clara	9.0	0-0.13	0-0.16	0-0.29
Ballona	2.6	0-0.43	0.47	0.47-0.90
Los Angeles	9.0	1.2-1.9	0.9-1.1	2.1-3.0
Santa Ana	1.0	ND	0.11-0.23	0.11-0.23

[a] Accumulated storm flow from which samples were obtained for chlorinated hydrocarbon analysis.

Table 3. Flow-weighted mean concentrations ($\mu g/l$) of chlorinated hydrocarbons in Los Angeles River storm runoff, 1971-73

Date	Total DDT	Dieldrin	1242 PCB	1254 PCB	Total PCB
4-7 Dec 72	0.66	0.12	0-1.1	0.51	0.5-1.6
27 Feb to 1 Mar 73	1.5	0.16	0-3.2	1.0	1.0-4.2
6-9 Mar 73	0.78	0.16	0-1.0	0.83	0.8-1.8
11-12 Mar 73	0.60	0.08	0-2.6	0.71	0.7-3.3
Average blank	<0.004	<0.001	<0.002	<0.001	<0.003
Averages					
1972-73	0.92	0.14	0-1.9	0.77	0.8-2.7
1971-72	0.93	0.16	1.2-1.9	0.9-1.1	2.1-3.0

The results indicate that the Los Angeles River carried an order of magnitude more PCB's via storm runoff than any other channel in the Bight. Further, the estimated input of 1254 PCB in storm runoff from the major channels in the Los Angeles Basin was 235 kg, constituting 86 to 98 percent of the estimated total storm runoff input of this contaminant to the Bight. These channels also carried more than 80 percent of the dry-weather input of 1254 PCB. A comparison of mass emission rates for all areas via the dry-weather and storm runoff for water year 1972-73 is presented in table 4. This summary illustrates that, although dry-weather flow constituted almost 20 percent of the total surface runoff volume during the year, it carried only about 5 percent of the measurable PCB's. Thus, it appears that storm runoff is the dominant mode for surface runoff inputs of PCB to the Bight, and that the Los Angeles Basin is the principal source region.

AERIAL FALLOUT

We conducted our aerial fallout surveys by exposing glass plates sprayed with mineral oil for about 1 week at 13 coastal and 5 island stations between Point Conception and the U.S./Mexico border. Two 13-week surveys were conducted in summer 1973 and spring 1974, and a 3-week survey was conducted during fall 1974. Although fallout rates of 1242 PCB were not obtainable, apparently because of poor retention of this relatively volatile mixture, the collection efficiency for 1254 PCB over 1-week exposure was determined to be about 50 percent (ref. 6).

During the first two surveys, net flux values for 1254 PCB generally ranged between 50 and 150 ng/sq m/day; values at certain coastal stations at the edge of the Los Angeles Basin were higher. During the third survey, however, values were higher by a factor of 2, on the average. The fall and winter seasons in southern California are characterized by desert (Santa Ana) winds, which occur periodically and transport the polluted air of the Basin out over the Bight. These data yielded seasonal fallout estimates for spring, summer, and fall of about 1,400, 1,430, and 2,700 kg/yr, respectively. Giving equal weight to each seasonal survey results in an estimated annual input of about 1,800 kg/yr 1254 PCB to the study area via dry aerial fallout. (Rainfall is so infrequent in this region that its overall effect is judged to be negligible.)

The estimated annual aerial inputs of 1254 PCB to the individual sectors of the study area are shown in

Table 4. Estimated 1972-73 mass emission rates of PCB via storm and dry-weather runoff to the Bight

Type	Volume (10^6 cu m)	PCB (kg/yr) 1242	PCB (kg/yr) 1254	PCB (kg/yr) Total
Storm	574	0-520	240-270	240-790
Dry weather	127	0-30	7-8	7-40
Total	700	0-550	250-280	250-830
% dry weather	18	2	7	3-5

figure 2. Highest values (each over 100 kg/yr) occurred in the five sectors off Los Angeles and Orange Counties (centering around the Palos Verdes Peninsula). This portion of the coastal plain of southern California is most affected by air pollution. The three sectors between Zuma Beach and Newport Beach (100 km by 50 km), which account for only about 10 percent of the 50,000-sq-km study area, received approximately 25 percent of the total measured PCB input. Thus, we find a pattern similar to that for surface runoff: The central basin area appears to be the single most important source of 1254 PCB transported via the atmosphere to the waters of the Bight.

DIRECT INDUSTRIAL DISCHARGE

Most of the industrial wastewaters released directly to marine waters off southern California occur in San Pedro and San Diego Harbors. These harbors also receive a wide variety of industrial discharges. Thus, we were able to study a number of different types of industrial effluents under similar conditions by surveying the two harbors (ref. 6). Estimates for overall industrial inputs of 1254 PCB to San Pedro Harbor were obtained by calculating flow-weighted concentrations in three types of effluents—power plant cooling water, fish cannery wastes, and "other industrial" discharges. The flow-weighted concentrations were then multiplied by the reported total flows of each of the three classes of industrial wastewaters into San Pedro Harbor to obtain estimates of annual mass emission rates. The values are presented in table 5. Corresponding data for San Diego Harbor are given in table 6.

These data provide no indication of significant release of PCB's to the harbors from the industrial effluents studied. However it should be noted that shipyard-related discharges into Los Angeles Harbor had the highest 1254 PCB concentrations of the industrial discharges surveyed; vessel cooling water, ballast effluent, and oil tanker clean-down water contained 0.6, 1.5, and 2.1

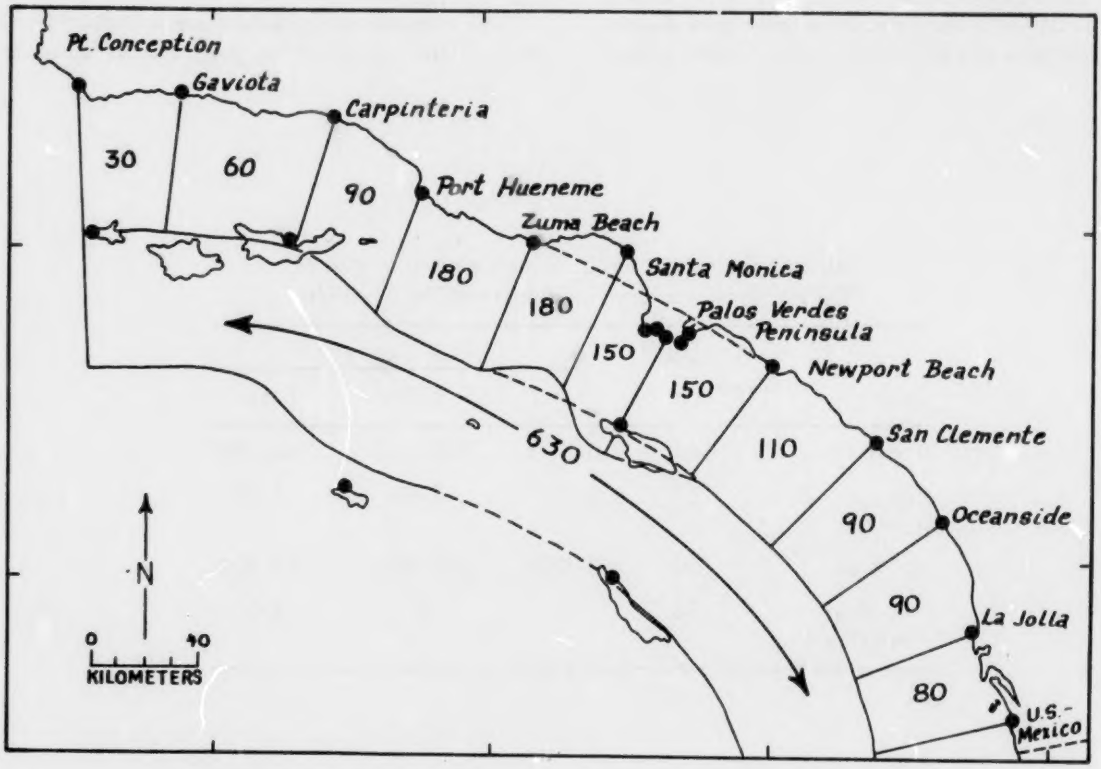

Figure 2. Estimated annual input of 1254 PCB (kg/yr) to sectors of the Southern California Bight via dry aerial fallout during 1973-74.

Table 5. Flow-weighted mean concentrations and estimated 1973 mass emission rates of 1254 PCB in industrial discharges to San Pedro Harbor

Discharge type	Total flow (mgd)	Concentration (μg/l)	Mass emission rate (kg/yr)
Power plant cooling water	1,020	0.01	14
Fish cannery wastes	15	0.09	2
Other industrial wastes	250	0.10	35
Total			50

Table 6. Flow-weighted mean concentrations and estimated 1974 mass emission rates of PCB in industrial discharges to San Diego Harbor

		1242 PCB		1254 PCB	
Discharge type	Total flow (mgd)	Concentration (μg/l)	Mass emission rate (kg/yr)	Concentration (μg/l)	Mass emission rate (kg/yr)
Cooling waters	757	<0.03	<30	0.01	10
Brine waters	0.5	<0.01	<0.01	0.05	0.03
Flume waters	2.8	<0.02	<0.07	0.01	0.05
Total			<30		10

μg/l 1254 PCB respectively. These relatively high levels are consistent with our observations that highest levels of this contaminant in harbor mussels occurred in regions of greatest vessel activity (ref. 8-11).

VESSEL ANTIFOULING PAINT

In 1973, we surveyed numerous drydock or haulout facilities in southern California harbors and marinas (ref. 12). Our objective was to obtain a representative picture of antifouling paint usage in southern California. In only 7 of the 28 wet paint samples analyzed, 1242 PCB or 1254 PCB were detected; levels were generally on the order of 1 mg/l or below. Although two samples had total PCB concentrations of approximately 40 mg/l, median values for 1242 and 1254 PCB were 0.3 mg/l and 0.7 mg/l, respectively. Combination of these median values with the estimated 300,000 liters of antifouling paint applied annually to recreational, commercial, and naval vessels in marinas and harbors of the Bight indicates that marine inputs of PCB from this source now are completely negligible.

However, four samples of old paint scraped from vessel bottoms yielded total PCB concentrations of 270, 3,300, 56,000, and 150,000 ppm, respectively. Furthermore, the two highest concentrations (approximately 5 and 15 percent on a dry weight basis) correspond with an observation by Dr. V. McClure,* who found that a paint chip collected in a zooplankton trawl in 1970 contained approximately 10 percent PCB. Polychlorinated biphenyls are reported to have been used extensively as a plasticizer in paints before such use was banned in this country in 1971. Our survey revealed that, on the average, the density of antifouling paints used in southern California is about 1.5 dry kg/l. Therefore, if such paints did contain 10 percent PCB in the past, an application rate of 300,000 l/yr would correspond to a PCB usage of 45,000 kg/yr. Antifouling paints are designed to slough off with time, and an estimated 5 to 10 percent of the old paint removed from vessel bottoms is believed to be carried back to the harbor waters (ref. 13). Thus these observations point to the possible importance of antifouling paints in the past as a source of PCB's to the coastal marine environment.

HARBOR FLUSHING

In view of the relatively high levels of PCB contamination found in southern California harbors (refs. 8-11), we attempted to determine if any significant levels of PCB's could be detected in the waters moving from such harbors into the open ocean during periods of peak tidal flow (ref. 6). Figure 3 illustrates the results obtained from the San Diego Harbor survey; net concentrations of 1254 PCB for the replicate samples of each collection period are plotted against tidal flow. Although there is some evidence of increased PCB concentrations near the time of tide reversal, when the greatest influence of "back harbor" water would occur near the harbor mouth, the measured concentrations were extremely low; flow-weighted mean concentrations for San Pedro† and San Diego Harbors were 1.5 and 1.8 ng/l, respectively.

By combining the measured concentrations with tidal flow values obtained from the current meter measurements and channel geometrics, we obtained estimates for maximum net transport of 1254 PCB from the harbors to the adjacent coastal ecosystem. Because of the extremely low concentrations observed, none of the

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*National Marine Fisheries Service, La Jolla, personal communication.

†Located just east of Palos Verdes Peninsula (figure 2).

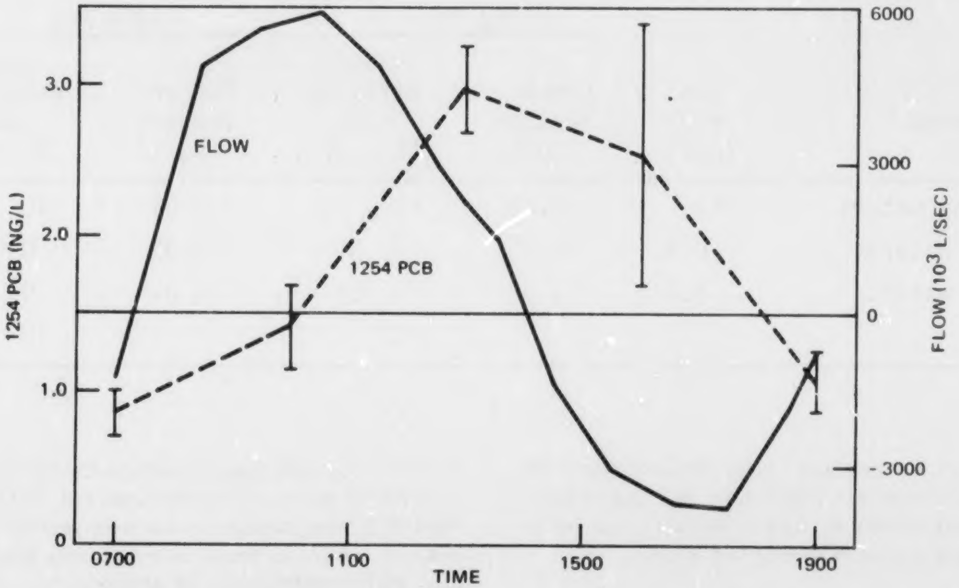

Figure 3. Concentrations of 1254 PCB in surface seawater collected over a semidiurnal tidal cycle at the mouth of San Diego Harbor, November 12, 1974. Positive flow represents outflowing water; negative, inflowing. Vertical bars indicate individual replicate values.

values exceeded 50 kg/yr when extrapolated to an annual basis. Thus, these pilot seawater surveys have provided no indication that any of the three harbors is now a significant source of polychlorinated biphenyls to the coastal marine ecosystem.

OCEAN CURRENT ADVECTION

As a part of this inputs study, we attempted to estimate the quantities of measurable chlorinated hydrocarbons in surface seawater annually flowing through the Southern California Bight. In October 1973, we sampled surface seawater at seven stations along a north/south transect (120°35′W) at the northwestern edge of the Bight (ref. 6). The transect began off Point Conception and ended approximately 300 km west of Oceanside. Circulation patterns (ref. 14) indicated that the transect chosen should have intersected California Current water flowing into the Bight.

Replicate 40-liter samples were collected in a stainless steel bucket, which was lowered from the bow of a research vessel that was slowly underway. Immediately upon collection, the replicate samples were passed through polyurethane foam columns and taken to Bodega Marine Laboratory for analysis. The results indicated average concentrations for total DDT and 1254 PCB of 0.7 and 0.4 ng/l, respectively.

These data may be used to obtain rough estimates of the quantity of these synthetic organics carried into the Bight via ocean current advection. Taking the length of the Bight as 500 km and the width influenced by the coast as 100 km, the effective study area is 5×10^{10} sq m. Assuming the mixed surface layer to be 50 m, the volume of the mixed layer is 2.5×10^{12} cu m. It has been shown that the mean residence time of water in this mixed layer is on the order of 3 months (ref. 14). Thus, the estimated advective flow rate of California Current water through the Southern California Bight is 10^{13} cu m/yr, equal to 10^{16} l/yr. Corresponding estimates for advective flux rates of measurable DDT and 1254 PCB compounds are 7,000 and 4,000 kg/yr, respectively.

CONCLUSIONS

Table 7 summarizes input rates of polychlorinated

Table 7. Inputs of PCB's to the Southern California Bight

Route	Year	PCB's (kg/yr)		
		1242	1254	Total
Municipal wastewater	1974	4,300	1,100	5,400
Surface runoff	1972-73	≤ 550	250	≤ 800
Aerial fallout	1973-74	-	1,800	-
Industrial discharges	1973-74	< 180[a]	60	< 250
Antifouling paint	1973	< 1	< 1	< 1
Harbor flushing[b]	1974	-	< 150	-
Ocean currents	1973	-	4,000	-

[a] Assuming that the maximum 1242/1254 PCB ratio (3:1) found in San Diego Harbor also applies to San Pedro Harbor industrial discharges.

[b] San Pedro, Newport, and San Diego Harbors.

biphenyls to the marine waters off southern California. These data indicate that, in recent years, municipal wastewaters have been the dominant source of PCB's to this coastal marine ecosystem. In 1974, more than 5 metric tons of this synthetic organic material were discharged via the five major treatment plants in the Bight; the fact that the 1254 PCB values for municipal wastewaters and ocean current transport were of the same order of magnitude testify to the importance of the wastewater input route.

Aerial fallout is the other important route by which PCB's enter the waters of the Bight. The 1973-74 estimate of 1,800 kg for 1254 PCB exceeded the 1974 input from municipal wastewaters (1,100 kg), and the fact that PCB levels in municipal wastewater are continuing to decrease suggests that aerial transport of these contaminants may become the dominant input mode in the future.

Finally, these studies have demonstrated that, in southern California, surface runoff is only a secondary PCB source, while contributions from industrial discharges and antifouling paint use in the major harbors appear to be insignificant. Although intertidal mussels in these harbors had an order of magnitude more PCB in their soft tissues than did nearby coastal specimens, we found no evidence that harbor flushing constitutes a significant source of polychlorinated biphenyls to the coastal marine ecosystem.

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PCB CONTAMINATION OF SOUTHERN CALIFORNIA MARINE ORGANISMS

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and Theadore C. Heesen*

Abstract

In the past, the submarine discharge of municipal wastewater has been the dominant source of PCB to the marine environment off southern California. However, the level of input from this source has steadily decreased during the period 1972 (> 19,000 kg/yr) to 1975 (2,600 kg/yr). Despite the significant decrease in this input, no significant decrease was observed over the 3-year period 1971-72 to 1974-75 in the level of PCB in muscle tissue of Dover sole, Microstomus pacificus, *collected from the major municipal wastewater discharge sites. In contrast, similar surveys of the intertidal mussel,* Mytilus californianus, *indicated that the PCB levels in this marine organism had significantly decreased over the same time period.*

Bight-wide surveys of M. californianus *and the yellow rock crab,* Cancer anthonyi, *indicate that PCB contamination of these marine animals is low but widespread. Levels of PCB in the harbor mussel,* M. edulis, *were highest in specimens collected from areas of intense vessel activity. A convenient and effective system for continuously monitoring PCB levels in the marine environment is described.*

INTRODUCTION

Over the last 4 years, the Southern California Coastal Water Research Project has been studying the inputs of PCB's to the Southern California Bight. A number of sources of PCB's (most closely resembling Aroclors 1242 and 1254) to the marine environment off southern California have been identified and their inputs quantified. The submarine discharge of municipal wastewater has been the dominant source of PCB to the marine environment off southern California. However, the level of input from this source has steadily decreased during the period 1972 (> 19,000 kg/yr) to 1975 (2,600 kg/yr). Dry aerial fallout also appears to be an important source, and if wastewater inputs continue to decrease, fallout may become the dominant input mode of the future. PCB inputs from surface runoff are of secondary importance, and those from direct industrial discharges and vessel antifouling paints are relatively insignificant. Detailed results of our inputs studies are presented elsewhere in these proceedings (ref. 1) and in a background paper (ref. 2).

Other workers have studied PCB contamination off California and elsewhere in the United States in the past decade. During the summer of 1975, several reports of high PCB levels in fish collected from Lake Michigan and the Hudson River caused public concern about PCB residues in commercial fish and sport fish (refs. 3-6). Munson's work in California (ref. 7) indicated that, in 1972, concentrations of these chlorinated hydrocarbons were low but widespread in the southern California marine community.

The work of Allen and his colleagues (ref. 8) with rhesus monkeys attests to the toxic effects of short-term, low-level (25 ppm) exposures to PCB's on non-human primates. Later results of their experiments on the monkeys showed that exposure to PCB's at the 2.5- and 5-ppm levels was related to spontaneous abortions and the birth of undersized infants (ref. 9). Also, de Long et al. (ref. 10) found PCB's to be associated with premature births in the California sea lion.

In conjunction with our studies of PCB sources, we undertook research into the fates and effects of PCB's in marine animals. Our research involved several organisms common locally (the Dover sole, *Microstomus pacificus*, a benthic flatfish; the yellow rock crab, *Cancer anthonyi*; and an open coast and a harbor mussel, *Mytilus californianus* and *M. edulis*, respectively), and three distinct programs:

- Regional surveys of PCB levels in the flatfish, the crab, and the mussels. Specimens from stations throughout the Southern California Bight (fig. 1) were analyzed to determine the levels of PCB's present in their tissues and plot the distribution of the substances in the Bight. Changes over time were noted, as were relationships between the concentration levels and man's centers of activity along the coast (wastewater discharge, major harbors). We also sought to identify the dominant PCB

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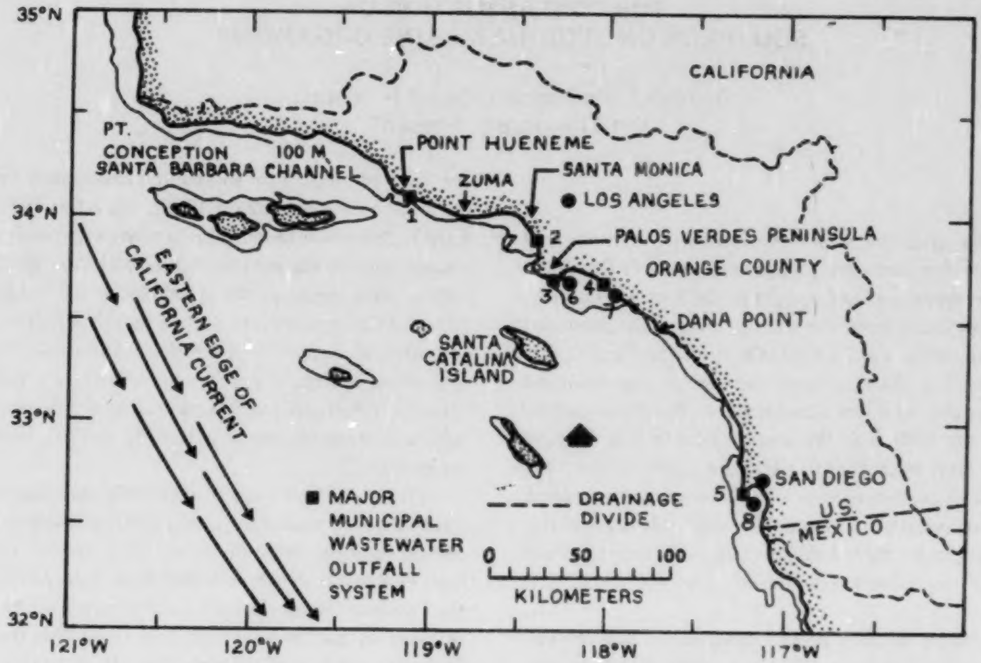

Figure 1. The Southern California Bight. Outfall systems are (1) Oxnard City, (2) Hyperion, Los Angeles City, (3) Whites Point, Los Angeles County, (4) Orange County, and (5) San Diego City. Major harbors are (6) San Pedro, (7) Newport, and (8) San Diego.

present by determining if the PCB's found most closely resembled Aroclor 1242 or Aroclor 1254.* Finally, we investigated the PCB levels in specimens of the flatfish afflicted with a fin erosion disease prevalent around several southern California wastewater outfalls.

- A study of the relative amounts of PCB's in the various tissues of the flatfish and the crab.
- The design of a convenient and effective system for continuously monitoring PCB levels in the marine environment.

This paper presents a brief summary of the results of these studies.

SAMPLING PROCEDURES AND CHEMICAL ANALYSIS

McDermott et al. (ref. 11) report in detail the procedures used for the collection and preparation of biological samples as well as the subsequent PCB analyses by electron-capture gas chromatography for all research programs discussed in this paper.

RESULTS AND DISCUSSION

Regional Surveys of PCB's in Marine Animals

Two Bight-wide surveys of PCB contamination in the benthic flatfish, Dover sole, were conducted during 1971-72 and 1974-75 (ref. 11). The results of these regional surveys (table 1) revealed that PCB levels in specimens collected near the three largest municipal wastewater discharge sites off Palos Verdes, Santa Monica, and Orange County were significantly higher than levels found in specimens from regions with little or no wastewater discharge. Despite the high levels of PCB in the fish from the outfall areas, less than 2 percent of all Dover sole taken in the two surveys had muscle tissue PCB concentrations that exceeded the Federal Food and Drug Administration's tolerance of 5 mg/wet kg in the edible portion of fish intended for interstate commerce.

Over the 3-year study period, municipal wastewater

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*Throughout the text, 1242 PCB and 1254 PCB have been used to distinguish between substances most closely resembling Aroclor 1242 and Aroclor 1254, respectively.

Table 1. Total PCB concentrations (mg/wet kg) in muscle tissue of Dover sole *Microstomus pacificus.*[a]

	1971-72 (n^b = 110)		1974-75 (n^c = 165)	
Region	Region median	Station range	Region median	Station range
Point Hueneme	0.1	0.1	0.06	0.05-0.07
Santa Monica	1.5	0.4-2.8	2.0	1.0 -2.3
Palos Verdes	1.9	1.1-6.3	1.3	0.06-2.5
Orange County	0.7	0.1-1.2	0.6	0.3 -2.8
Dana Point	0.06	0.03-0.09	0.7	0.03-0.14
San Diego	-	-	0.2	0.09-0.6
Santa Catalina Island	0.04	0.03-0.04	0.05	0.03-0.07

[a] After ref.11. There is no statistically significant difference between the 1971-72 and the 1974-75 levels.

[b] Composite samples of Dover sole muscle tissue.

[c] Individual samples of Dover sole muscle tissue.

emissions of total PCB decreased (ref. 1). Yet we observed no statistically significant decrease in the levels of total PCB in the Dover sole. This finding indicates that other factors or inputs of these chlorinated hydrocarbons are involved in maintaining the total PCB levels in this fish.

During 1971 and 1974, Bight-wide surveys of the open coast mussel were also conducted (refs. 11,12). The results are shown in table 2. A regional survey of the yellow rock crab was also conducted during 1971 (ref. 11). Table 3 presents the median concentration of total PCB in the muscle tissue of the crabs collected in each region. These surveys indicate that the PCB contamination levels in these animals are low but widespread. Levels of total PCB in specimens near the major outfall systems were generally 10 to 100 times higher than levels in specimens collected from coastal or island control sites; however, the Federal tolerance level for PCB compounds was not exceeded in the edible portion of any specimens of these two species. The two surveys of *M. californianus* indicate that the level of PCB contamination in these mussels had decreased significantly over the 3-year interval, 1971-74, with a median percent decrease of 54 percent.

The dominant PCB observed in the three species surveyed was 1254 PCB; the other PCB identified was 1242 PCB. The percent composition of total PCB in the muscle tissue samples from the crab and mussel specimens was similar to that in the Dover sole muscle samples. The median 1974-75 composition of total PCB in muscle tissue of Dover sole taken near the five major discharge sites was 67 percent 1254 PCB and 33 percent 1242 PCB. This contrasted with the median 1974 composition of municipal wastewater emission of PCB: 29 percent 1254 PCB and 71 percent 1242 PCB. These data, along with a comparison of total PCB levels in Dover sole muscle tissue, surface sediments, and municipal wastewater emissions (table 4), indicate that the PCB levels in Dover sole are not dependent only upon the level of PCB discharged in wastewaters and that other factors, such as the solubility, volatility, the sediment load, or the relative biological uptake and

Table 2. Concentrations (mg/wet kg) of the PCB most closely resembling Aroclor 1254 in the whole soft tissues of *Mytilus californianus*.[a]

Station	1971	1974
Coastal		
Gaviota	0.05	0.01
Santa Barbara	0.03	0.02
Port Hueneme	0.12	0.37
Point Dume	0.08	0.04
Palos Verdes Peninsula		
Point Vicente	0.38	0.10
Royal Palm	0.52	0.14
San Clemente	0.05	0.02
Point Loma	0.12	0.06
Island		
Anacapa	0.01	0.01
Santa Barbara	0.07	0.02
Santa Catalina	0.02	0.01
San Nicolas	0.02	0.01
San Clemente	0.02	0.02

[a] After refs. 11, 12.

Table 3. Regional survey of total PCB concentrations (mg/wet kg) in the muscle tissue of individual yellow rock crabs, *Cancer anthonyi*, 1971-72.[a]

Region	No. of samples	Region median	Station range
Santa Monica	12	0.68	0.13 -2.2
Palos Verdes	16	0.82	0.30 -1.8
Outside San Pedro Harbor	5	0.49	0.32 -0.83
Orange County	9	0.32	0.23 -0.54
Ensenada[b]	3	0.014	0.012-0.018

[a] After ref. 11.

[b] Three chelae (*Cancer* species) purchased in fish market.

Table 4. Comparison of total PCB levels in muscle tissue of Dover sole, *Microstomus pacificus,* and bottom surface sediments and PCB mass emission rates in municipal wastewater.[a]

	1971-72			1974-75	
Region	Dover sole (mg/wet kg)	Sediments (mg/dry kg)	Municipal waste (kg/yr)	Dover sole (mg/wet kg)	Municipal waste (kg/yr)
Palos Verdes	1.9	3.6	11,600	1.3	1,270
Santa Monica	1.5	0.53	1,920	2.0	956
Orange County	0.7	0.02	5,800	0.6	2,100
San Diego	-	0.01	118	0.2	1,050
Point Hueneme	0.1	0.004	3	0.1	5

[a] After refs. 1,2,11.

retention rate of a given PCB are involved in the resulting levels of PCB's in this marine animal.

Distribution of PCB's in Tissues of a Benthic Flatfish and Crab

In February 1975, special collections of Dover sole were made off Palos Verdes and Orange County for the analysis of PCB's in the muscle, liver, gonad, heart, and kidney tissues (ref. 11). The results are presented in table 5. The level of PCB in the liver tissue was 10 to 20 times higher than the levels observed in the other tissues analyzed. To determine the liver and muscle tissue burdens of the two sets of fish, the median concentrations of PCB in the specific tissue was applied to the average wet weight of that tissue:

	Liver burden	Muscle burden	Ratio, liver to muscle
Palos Verdes	0.04 mg	0.02 mg	2
Orange County	0.02 mg	0.03 mg	0.7

The liver-to-muscle tissue burden ratio for PCB in Palos Verdes Dover sole was 2; for Orange County specimens, the ratio was 0.7. There are two possible explanations for the high liver-to-muscle tissue burden ratio in the Palos Verdes fish. A review of the literature indicates that (1) in metabolic disturbances, the lipid (fat) content in liver tissue may significantly increase, and (2) chlorinated hydrocarbons, which are hepatic poisons, induce fatty livers. As chlorinated hydrocarbons are associated with lipids, if either one or both of these situations had occurred in the Palos Verdes Dover sole, it would have resulted in an increase in the total PCB levels in the liver and a higher liver-to-muscle tissue burden ratio. The lipid content of these tissues is being studied, and further work on the physiological and ecological implications of these tissue burdens is underway.

Analysis of the PCB concentrations in the reproductive organs of *C. anthonyi* indicate that the gonadal tissues generally contain PCB levels five to ten times higher than the muscle tissue levels (ref. 11). Also, in females, the gonad had an absolute tissue burden of PCB that was 25 percent higher than the muscle tissue burden (see table 6).

As the reproductive organs of this crustacean are exposed to considerably higher levels of this synthetic organic than is the muscle, spawning may represent a significant distribution mechanism of these contaminants in the marine environment, and a significant factor in the elimination of a large percentage of the body burden of these materials from the crab. The long-term effects of PCB concentrations on reproduction or other biological processes in this crustacean are unknown.

Fin Erosion and PCB Levels in a Benthic Flatfish

The Dover sole collected off Palos Verdes are frequently affected by fin erosion; the disease has recently also become prevalent off Orange County. Data on specimens with eroded fins and those with healthy fins taken in 1974 in single trawls were paired. Utilizing the

Table 5. Total PCB concentrations (mg/wet kg) in composite samples of tissues of Dover sole, *Microstomus pacificus*, 1975[a]

Tissue	Palos Verdes		Orange County	
	Median	Range	Median	Range
Muscle	0.7	0.1-1.1	1.1	0.6-1.4
Liver	15	11-18	8.3	4.8-13
Gonads	1.4	0.8-5.2	0.8	0.7-4.6
Heart	1.8	1.5-3.2	0.5	0.4-0.8
Kidney	0.8	0.6-1.1	0.6	0.6-0.8

[a] After ref. 11.

Table 6. Analysis of PCB concentrations in the reproductive organs of *Cancer anthonyi*

	Male	Female
Percent of combined gonad/muscle tissue weight		
Gonad	7	27
Muscle	93	73
Tissue burden (mg)		
Gonad	0.009	0.031
Muscle	0.025	0.025

Wilcoxon signed-rank test, the levels of PCB in the muscle tissue of the unaffected fish and the diseased fish were found to be different at the 90 percent confidence level ($p = 0.10$). Although this level is not considered to be statistically significant, it shows a strong tendency for the total PCB levels to be higher in the diseased fish. The median values for the diseased and unaffected groups were 2 and 1 mg/wet kg, respectively (refs. 11,13).

If this association was dependent upon the input level of PCB, one would expect fin erosion to be dominant off Orange County where the input level of PCB is the greatest. One would also expect to find it off Santa Monica and San Diego where the level of input is similar to Palos Verdes. However, this is not the case. There are several possible reasons for the association between high PCB levels and fin erosion. The disease is predominantly found in the Palos Verdes region: Palos Verdes sediments have the highest levels of PCB in the Bight, thus contaminated sediments could be the dominant factor. Also, PCB—in combination with other constituents present in this region (DDT, hydrogen sulfide, trace metals)—could be involved in the development of the disease. It is also possible that PCB uptake is enhanced in diseased fish; hence the higher levels could be the result of the disease rather than a cause. These relationships are being studied further.

Surveys of PCB's in Harbor Mussel

Composite samples of the whole soft tissues of *Mytilus edulis* collected from three major southern California harbors (San Pedro, Newport, and San Diego) were analyzed for PCB's (ref. 11).

The survey showed that the harbor mussels had contamination levels up to 20 times those found in specimens of the same species collected from nearby coastal sites. Highest levels were found in mussels taken near regions of heavy vessel activity. Figure 2 shows the data for San Diego Bay, where the range of total PCB values near the commercial docks and navy moorings (0.80 to 1.3 mg/wet kg) is three to four times higher than the values observed at other inner harbor sites, and ten times greater than levels in nearby coastal mussels.

The levels of 1254 PCB in seawater at the mouths of the harbors were generally on the order of 1 part per trillion (ref. 1,2). The values of 1254 PCB in the whole soft tissues of the harbor mussels were 100,000 times these seawater levels.

Although most antifouling paints presently applied to vessel bottoms in southern California contain PCB concentrations of less than 1 mg/dry kg, a few samples of pre-1970 paint chips averaged about 10 percent PCB or 100,000 mg/dry kg (ref. 14). Thus it is possible that thousands of kilograms of this synthetic material could have been released annually to the harbor and coastal marine ecosystems, before the widespread use of non-recoverable PCB's was discontinued in the United States in the early 1970's.

Bouy Mussels: An Offshore Biomonitoring System

In June 1974, 4- to 6-cm long specimens of *M. californianus* were collected from Point Sal, an area known to be relatively free of PCB contamination. Within 1 day of collection, these mussels were transported to a taut-line bouy anchored off Whites Point, where wastewaters from Los Angeles County's Joint Water Pollution Control Plant discharged (ref. 11). The mussels were placed in net bags fastened at five levels between the sea surface (0.5 m) and the bottom sediments (35 m), which are highly contaminated with trace metals and chlorinated hydrocarbons at this site (refs. 15-17).

The mussels transported to the bouy system off Palos Verdes appeared to survive well under the test conditions: Less than 10 percent mortality was observed at any of the five levels (0.5 to 35 m) at which the mussels were suspended during the 3-month study.

On the average, mussels living at Royal Palm Beach, inshore of the buoy, contained 17 times as much PCB as the control specimens at the time of their transfer to the buoy northwest of the outfalls. Thus, exposure of these mussels to PCB was greatly increased upon their transfer to the discharge region.

The results indicated that there was a direct relationship between uptake of PCB and proximity of the bioindicator to the contaminated bottom sediments and to the wastewater plume, which is trapped beneath the thermocline. The bottom specimens became approximately 10 times as contaminated as did the surface specimens.

At the end of the 13-week period, the 1254 PCB concentrations in the whole soft tissues of specimens collected from Level 5 (closest to the bottom sediments) were approaching 0.4 mg/wet kg; those specimens from Level 1 (at the surface) were at 0.04 mg/wet kg. To date, the highest 1254 PCB concentration measured in the water above the outfalls is 4 ng/1 (ref. 12). This suggests a concentration factor on the order of 100,000 for 1254 PCB in the whole soft tissues of *M. californianus*, a number in good agreement with the estimate for *M. edulis* in the harbors.

SUMMARY

1. No statistically significant decrease in the Bight-wide levels of PCB in the muscle tissue of Dover sole was observed over the 3-year period 1971-72 to 1974-75.
2. The level of PCB contamination of the open coast mussel decreased significantly over the 3-year interval, 1971-74, with a median percent decrease of 54 percent.
3. Regional surveys of the yellow rock crab and open coast mussel indicate that PCB contamination levels in these animals are low but widespread.
4. For all three species surveyed, PCB levels in specimens collected near major outfall systems were generally 10 to 100 times higher than levels in specimens collected from coastal or island control sites.
5. The dominant PCB observed in the three species surveyed most closely resembled Aroclor 1254; the other PCB identified most closely resembled Aroclor 1242. In contrast, the dominant PCB discharged via municipal wastewaters most closely resembled Aroclor 1242.
6. The liver tissue of Dover sole generally had concentrations of PCB's 10 to 20 times greater than the levels found in the muscle, gonad, heart, and kidney tissues.
7. The liver-to-muscle tissue burden ratio for total PCB in Palos Verdes Dover sole was three times higher than the ratio for Orange County specimens.
8. The reproductive organs of the yellow rock crab generally contain PCB levels five to ten times higher than the muscle tissue levels.

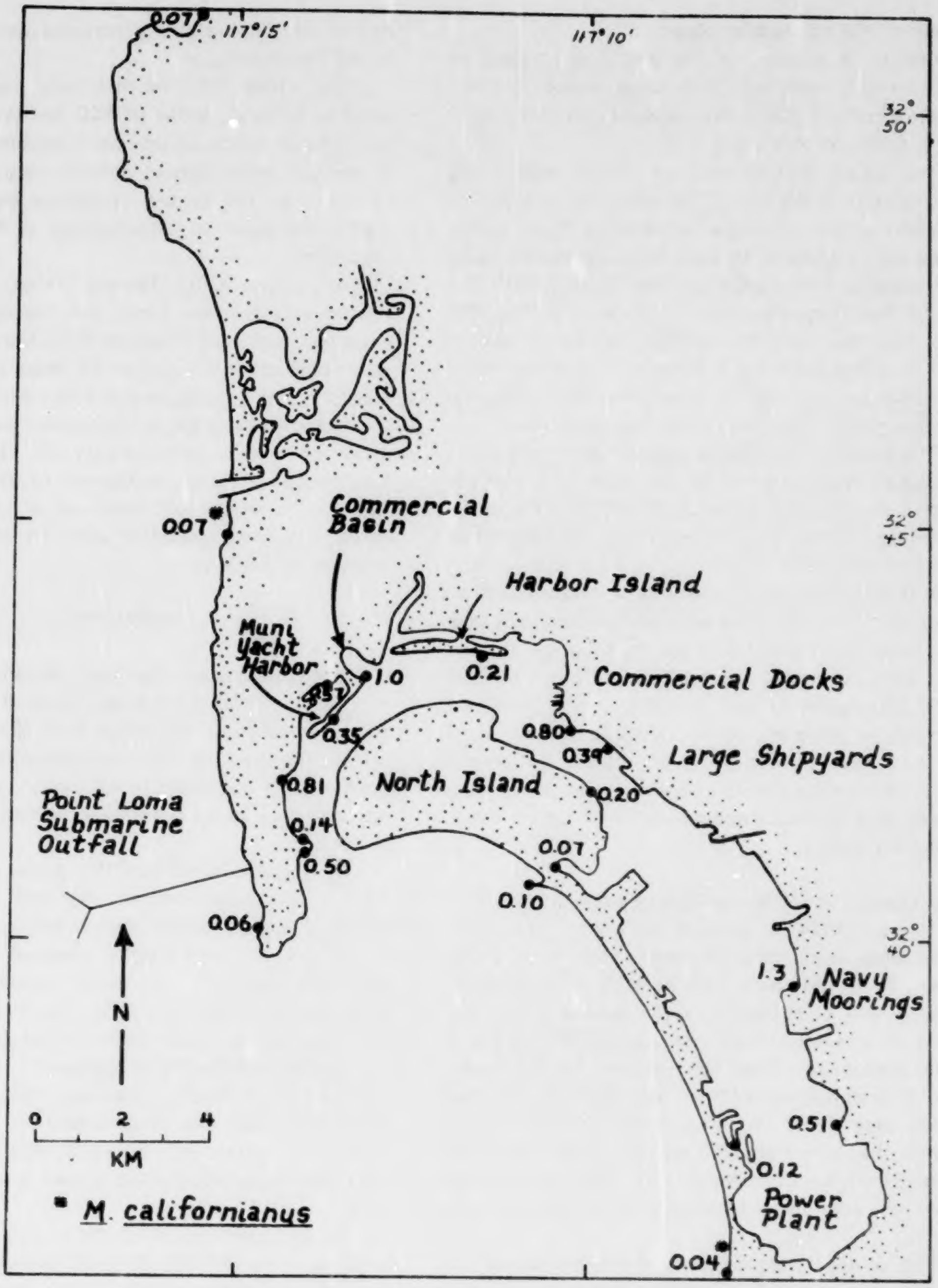

Figure 2. Total PCB concentrations (mg/wet kg) in whole soft tissues of *Mytilus edulis* in San Diego Bay, January 1974. (After ref. 11).

9. PCB's were found to be associated with the fin erosion disease prevalent in Dover sole collected around major municipal wastewater discharge sites at the 90 percent confidence level ($p = 0.10$).

10. Harbor mussels had PCB contamination levels up to 20 times those found in specimens of the same species collected from nearby coastal sites. Highest levels were found in mussels taken near regions of heavy vessel activity.

11. Although the mussel used in the buoy system was an intertidal organism, less than 10 percent mortality was observed at depths to 35 m. This mussel's hardiness, its ubiquitous distribution along many coastlines around the world, its very high ability for concentrating chlorinated hydrocarbons above seawater values, and its apparent ability to rapidly respond to changes in environmental levels of such contaminants make it a very useful bioindicator, both in natural intertidal communities and on offshore substrates.

The widespread use of nonrecoverable PCB's was curtailed in the United States during the early 1970's. However, the inputs of these persistent substances are still diffuse and difficult to control. There is a need for more knowledge of the effects of these materials in the marine environment; in particular, uptake rates, persistence, and biological effects should be studied.

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TRANSPORT OF CHLORINATED HYDROCARBONS IN THE UPPER CHESAPEAKE BAY

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Abstract

Under contract to the Maryland State Department of Natural Resources, the staff of the Westinghouse Ocean Research laboratory (together with subcontractors at Johns Hopkins University and the University of Maryland) recently completed the Upper Bay Survey—a program having as the central task the study of the routes, rates, sources, sinks, and reservoirs of chlorinated hydrocarbons (CHC's) in the upper Chesapeake Bay. Data from the field portion of this program will be presented; in particular, the levels of PCB's, chlordane, and DDTR found in bottom sediments, suspended particulates, zooplankton, and shellfish will be summarized. The discussion will include the role of sediment deposits as traps for CHC's; suspended particulates as transport vehicles for CHC's; movement of CHC's from the suspended particulate reservoir into the biological system, particularly shellfish and zooplankton; and the sources of CHC's in the upper Chesapeake Bay.

INTRODUCTION

The concept of the Upper Bay Survey grew directly from the Chester River Study (ref. 1), a program of more limited scope, which was completed in the fall of 1972. An objective of that program was to provide the Maryland Department of Natural Resources with environmental and resource management information essential to the local shellfish industry. These studies were directed toward determining the source and fate of chlorinated hydrocarbons and selected trace metals in the waters and sediments of this estuary. The approach was multidisciplinary. The project as a whole was considered experimental, since it was to encompass many aspects of an estuarine ecosystem with the declared purpose of supporting the practical needs of natural resource managers. Thus, the Chester River Study was a program of focused, applied research rather than a more basic program to extend knowledge in estuarine sciences.

As a result of this program, it became clear that the upper Chesapeake Bay was the most likely source of sediment-borne materials considered potentially harmful to the living resources of the Chester River. An extension of the Chester River project encompassing the bay between the Severn and Susquehanna Rivers became the next logical step in assessing the impacts of man-made substances in the bay ecosystem. The Upper Bay Survey (funded by the Mayland Department of Natural Resources) was begun in December 1973—once again multidisciplinary, but also multiinstitutional with the participation of scientists from the University of Maryland and The Johns Hopkins University. A 12-month research program was implemented with the following five definitive objectives:

1. To determine concentrations, distributions, and sources for chlorinated hydrocarbons (CHC's) including pesticides and polychlorinated biphenyls (PCB'S), and to determine the nature of transport paths, mechanisms, and rates in the Chesapeake Bay's waters, sediments, and organisms.
2. To determine immediate and longer-term biological consequences of chlorinated hydrocarbons and PCB'S on commercially important species.
3. To determine the distribution of bacteria on sediments and suspended particles of the upper bay, and to perform toxicological studies of the combined effects of pesticides and bacteria on oysters.
4. To institute numerical models for projecting contaminant distribution relative to the sources and to develop interrelations to biological impacts resulting from changes in the upper Chesapeake Bay's input stresses.
5. To report results in a format convenient for resource management and in a format for computer storage, retrieval, and augmentation.

In this paper, the data resulting from the chlorinated hydrocarbon analyses of bottom sediments, suspended sediments, zooplankton, and shellfish will be presented and discussed.

METHODS

Sample Collection. The bottom sediments were collected as grab samples using either a Wildco-Eckman Bottom Sampler or a Ponar Grab Sampler. In the laboratory, a portion of the sample was retained for sediment grain-size and clay mineralogy analyses, and the remainder air-dried for about 1 week. The shellfish samples were collected with a modified bottom trawl or a Ponar Grab Sampler and frozen for later analysis.

Suspended particulate samples were collected from surface, midwater, and 0.5 m from the bottom at the sampling stations illustrated in figure 1. The suspended

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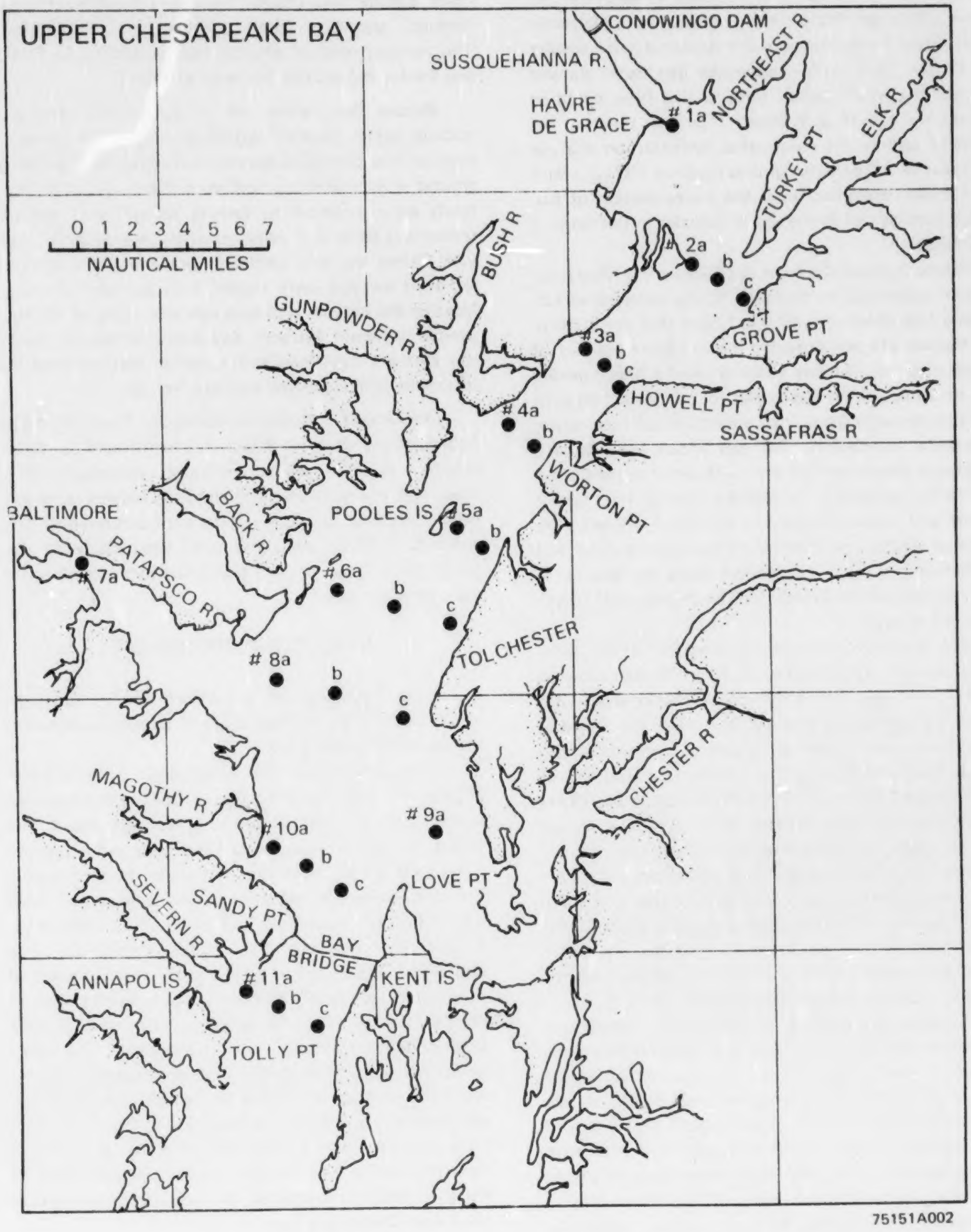

Figure 1. The principal stations at which samples and measurements were taken in the Upper Bay Survey.

particulates were collected by filtration through Gelman type-A glass-fiber filters having nominal pore diameters of 5-8 μ (the functional pore size appeared much smaller because our tests with Chesapeake Bay water showed that nothing which passed through this filter could be retained on a 0.45 μ Millipore filter). In order to get sufficient sample for chlorinated hydrocarbon analysis (in excess of 0.3 g dry weight) as much as 100 l of water was filtered depending upon the concentration of suspended particulates in the water (usually in the range, 3 to 30 mg/l).

Figure 2 shows the filter apparatus and a filter loaded with suspended particulates. It was determined empirically that filtering until two filters 14.5 cm in diameter became clogged provided the minimum amount of sample required. The bay water entered a ¾ inch garden hose suspended at the appropriate depth, passed through the filter, through the pump, and into a calibrated vessel for volume estimation. The wet filters were folded, wrapped in aluminum foil and transported to the laboratory. In the laboratory the samples were air-dried (about 2 days) and stored wrapped in foil until analysis. The weight of sample collected (and subsequently extracted) was determined by a calculation using the volume of water filtered and the concentration of suspended particulates in the water.

Two procedural restrictions prevented direct determination of the suspended particulate sample weights: first, the filters had to be preextracted with solvents to remove interfering substances and sealed in foil until use, which made preweighing undesirable; and second, had preweighing been done, the subsequent weights would have been dubious because these filters tend to shed and flake. At each point in time and space where a suspended particulate sample was collected for chlorianted hydrocarbon analysis, two suspended particulate samples were collected on 4.7 cm diameter Millipore or Nucleopore membrane filters (pore size 0.45 and 0.6 μ respectively)—one for determing the suspended particulate concentration and one for determining the suspended particulate grain-size diameters (ref. 2).

Zooplankton samples were collected as oblique tows with paired half-meter standard oceanographic nets using hexane rinsed 202-μ Nitex netting. A calibrated T.S.K.-type mechanical flowmeter was placed in the mouth of each of the two nets to record the volume of water passing through. Upon completion of each tow, the nets were carefully washed to concentrate the plankton in the cod end. After removal of the larger, gelatinous material by passage through a 4-mm sieve, the samples were concentrated with a 202-μ sieve. From each pair of net samples, one net collection was preserved for taxonomic and biomass enumerations, and the other sample was passed through a hexane-extracted Gelman glass-fiber filter to collect the zooplankton. The resultant pad of zooplankton (including the filter) was frozen and retained for later analysis.

Sample Preparation. All of the samples were extracted using Soxhlet extraction with 2:1 hexane-acetone (the biological samples were first dried by being ground with anhydrous sodium sulfate). All of the extracts were subjected to fuming sulfuric acid cleanup procedures (refs. 3,4) prior to gas chromatographic analysis. Before the acid treatment procedure, the bottom sediment extracts were treated wtih activated alumina. Most of the bottom sediment extracts, some of the suspended sediment extracts, and several of the zooplankton extracts contained sulfur, which was removed by treatment with elemental mercury (ref. 5).

Chlorinated Hydrocarbon Analysis. The chlorinated hydrocarbons were identified and quantitated by multicolumn, electron-capture, gas-liquid chromatography. Peak relative retention times and peak heights (or areas) were compared to those of standard compounds. The amounts of PCB's and chlorinated pesticides were estimated by a manual subtraction procedure correcting for the overlapping peaks.

RESULTS AND DISCUSSION

Table 1 summarizes the chlorinated hydrocarbon levels found in the various types of samples collected in the upper Chesapeake Bay.

The standard deviation values given in table 1 emphasize the fact that the range of CHC values observed within any particular sample type was very broad. The values for CHC's on suspended sediment and zooplankton varied greatly from one station to the next during the same collection period or at the same station from one collection period to the next. Apparently these values fluctuate rapidly enough (temporally and spatially) that only the unusually large events in terms of time and/or space will be observed at enough points to describe a trend when the sampling is limited to a fairly small number of samples, as in this program. The multidisciplinary approach of this program, however, circumvents this problem somewhat by providing many different types of data that are comparable in space and time. As will be seen below, these data then can be examined for interrelationships relevant to the understanding of the dynamics of chlorinated hydrocarbon movements in the upper Chesapeake Bay.

For the purposes of this discussion, the PCB's will be discussed as total PCB (the sum of the values reported as Aroclor 1242, 1248,1254, and 1262), and in some

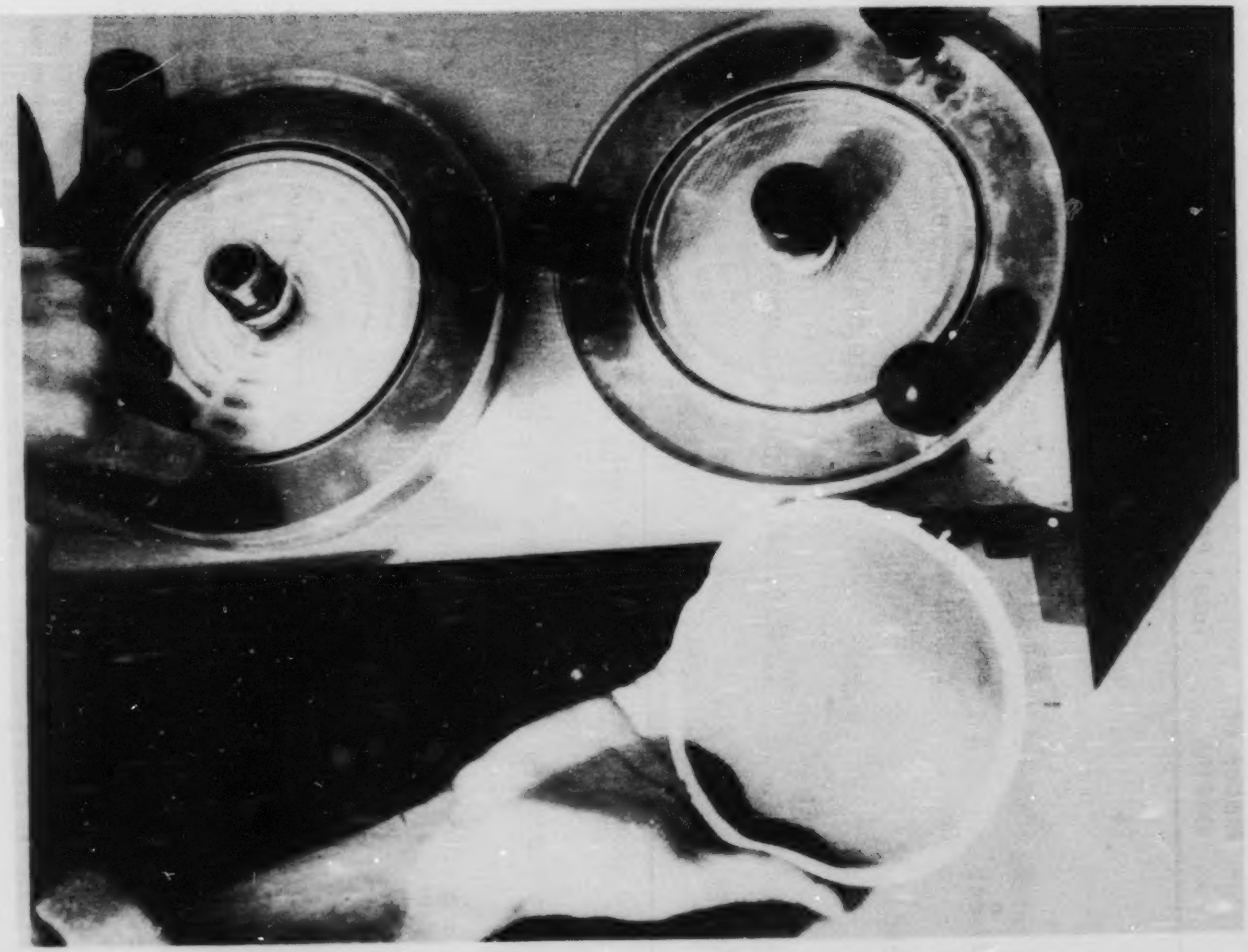

Figure 2. Filtering apparatus for collecting suspended sediment samples and a Gelman filter containing suspended sediments.

Table 1. Average chlorinated hydrocarbons found in the upper Chesapeake Bay (Standard deviations are in parentheses)

Sample type	Number of samples	Total PCB	Total chlordane	Total DDT
Shellfish (wet,ppm)[a]	26	0.052(0.037)	0.016(0.017)	0.035(0.041)
Plankton (wet,ppm)	70	0.50(1.4)	0.041(0.032)	0.16(0.68)
Suspended sediment (dry,ppm)[b]	66	0.92(0.87)	0.061(0.086)	0.057(0.066)
Bottom sediment (dry,ppm)	54	0.28(0.57)	0.0052(0.014)	0.051(0.067)
Plankton (H_2O,ppt)[c]	69	0.042(0.164)	0.0038(0.0083)	0.010(0.035)
Suspended sediment (H_2O,ppt)	68	12(14)	0.53(0.88)	0.78(1.5)

[a] The values are expressed as μg CHC found per g wet weight of material extracted.

[b] The values are expressed as μg CHC found per g dry weight of material extracted.

[c] The values are expressed as ng of CHC found per l of water filtered to collect the material extracted.

cases, all of the CHC's found will be summed and discussed as total CHC. The PCB residue pattern observed most often matched Aroclor 1254 or a mixture of Aroclors 1254 and 1262. (Aroclor 1242 or 1248 was found in many of the samples.)

By comparing the bottom sediment data with the suspended sediment (dry) data, one can see that the PCB and chlordane concentration are 4 to 10 times higher in the suspended sediments. The higher values in the suspended sediment probably occur for one, or most likely, both of the following reasons: the average grain-size of the suspended sediments is much smaller than that of the bottom sediments, resulting in a greater surface area for adsorption per unit weight; and although the suspended sediments are primarily inorganic materials, the phytoplankton, which were included in these samples, probably bioconcentrated chlorinated hydrocarbons to some extent.

If one accepts the above discussion, one is left with the dilemma that the DDTR does not appear higher in the suspended sediments as do the PCB and chlordane. But all uses of DDT were banned in the United States at the end of 1972. If this ban had the effect of decreasing the DDT levels flowing into the Chesapeake Bay, the concentrations of DDT residues in the suspended sediments would be less relative to those in the bottom sediments because the bottom sediment samples consist of materials deposited during the sampling year and previous years as well, while the suspended sediment samples consist primarily of materials that entered the bay during the sampling year. While this explanation is plausible, it seems somewhat suspicious that samples were taken fortuitously at just the right time to yield exactly the same DDT residue levels in both the suspended sediments and the bottom sediments.

The data in table 1 also show that the CHC's are bioconcentrated in passing from the suspended sediment into the zooplankton. To evaluate the magnification, one must first convert the plankton (wet) values to plankton (dry) to allow comparison with the suspended sediment (dry) values. If one conservatively uses 0.10 as the plankton dry weight to wet weight ratio (ref. 6), one then multiplies all of the plankton (wet) values by 10 to convert to plankton (dry) and then calculates the following bioconcentrations as the CHC's pass from the suspended sediments to the zooplankton: PCB, 5.4; chlordane, 6.7; and DDTR, 28. The DDTR value seems unreasonably high, and it probably is. If two anomalously high DDTR values are excluded from the total and the average is computed from the other 68 values, the average DDTR in plankton (wet) become 0.045, leading to a magnification of 7.9–a value more compatible with the others.

The CHC data for suspended sediments and zooplankton are expressed in two ways, as mentioned earlier, but further amplification should be made. Environmental residues of chlorinated hydrocarbons (chlorinated pesticides and polychlorinated biphenyls) usually are reported as parts per million (ppm) dry weight (or wet weight) based upon the dry weight (or wet weight) of sample which was extracted for the analysis. If the levels are very low, parts per billion (ppb) or parts per trillion (ppt) also are used. The data then represent the concentration of CHC's present in that sample. In the consideration of transport of CHC's (rates, routes, etc.) the amount of CHC being transported by a water movement frequently is of more interest than the concentration of the CHC's on the suspended sediments in the water.

Therefore, in addition to the data being presented in the usual fashion, the zooplankton and suspended sediment CHC data are presented as nanograms (10^{-9}g) of CHC on zooplankton or suspended sediment per liter of water (by definition, ppt). These values represent the amount of CHC associated with the suspended sediments or the zooplankton contained in a 1 liter sample of bay water. In a sense, the numbers can be compared directly because they have been normalized to a unit volume of water. For instance, referring to table 1, one finds the average PCB values in the bay water on suspended sediment and zooplankton to be 12 ppt and 0.042 ppt, respectively. On the average, a given volume of bay water will have about 300 times as much PCB in the suspended sediment fraction as in the zooplankton fraction.

Using these values, one also could calculate approximate biomagnification values from the shellfish data. The concentrating ability of shellfish usually is expressed as the level accumulated in the shellfish tissue (wet weight) divided by the exposure concentration in the water. Using the values for CHC in the water column on suspended sediment, the shellfish concentration factors are approximately: PCB, 4,000; chlordane, 30,000; and DDTR, 45,000. These values should be considered rough estimates, because the average values used for CHC's in the water column probably do not represent accurately the values at the water-bottom sediment interface inhabited by these shellfish.

Figure 3 is a log-log plot of total CHC concentration in the water column on suspended sediment (ppt) versus the concentration of suspended sediment in the water (mg/l). Although the data points are widely scattered, the data could best be enclosed in an oblong field with an upward tilt to its long axis. This indicates a positive relationship between the parameters plotted (if the parameters were unrelated–that is, varied independently–the long axis of the oblong would be either vertical or horizontal). The correlation obviously is not followed closely by all of the data, but in general, it indicates that the bay water samples which had high suspended sediment concentrations also had high levels of CHC's in the water on suspended sediments. If all suspended sediments in the water contained the same amounts of CHC's (ppm, dry weight), a perfect correlation would have been observed in figure 3. The variations in the concentrations of CHC's on suspended sediments are responsible for the scattering of the data.

Figure 4 is a log-log plot of total CHC's in the water on zooplankton (ppt) versus the zooplankton biomass in the water (mg/m^3). Although the data are somewhat scattered, there is an obvious positive relationship–as the zooplankton population in the water increases (increasing biomass), the amount of CHC's in the water on zooplankton increases. The existence of this relationship establishes that there is movement of CHC's into the aquatic food chains which include the zooplankton community. The fact that the zooplankton population contains only a small fraction of the CHC's present in the water column, considered with the relationship observed in figure 3, suggests that the movement of CHC's into the biological system (via zooplankton) from the nonbiological system (suspended sediments) is not influenced by changes in the concentration of suspended sediment but is regulated by whatever regulates the zooplankton population. One could visualize the suspended sediments in the water column as being a reservoir of CHC's, which flow into the biological system when zooplankton blooms occur.

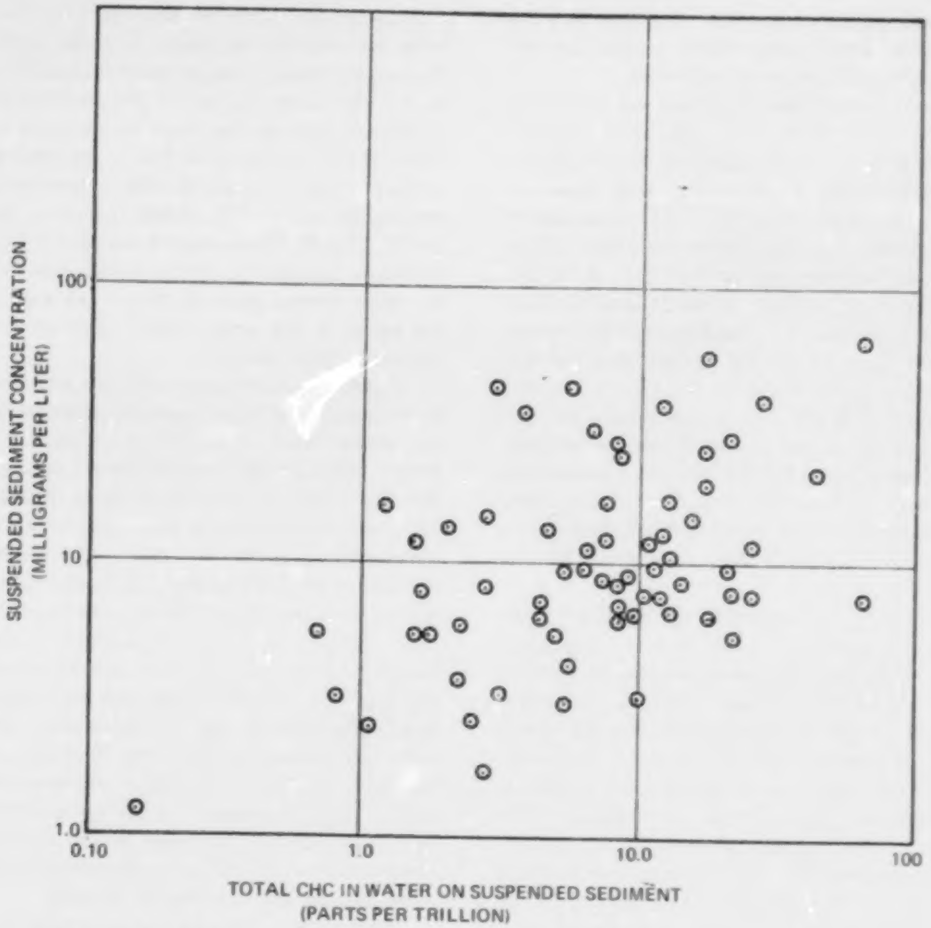

Figure 3. A plot of the log of the suspended sediment concentration in the water (milligrams per liter) versus the log of the total CHC concentration in the water associated with the suspended sediment (parts per trillion).

Obviously, this concept is an over simplification of a very complex system. More likely, the phytoplankton are important in the transport of CHC's from the non-biological reservoir into the zooplankton food-chain. Unfortunately, the sampling procedure for gathering suspended sediments involved pumping the water through a filter, so the phytoplankton were included in the fraction called suspended sediments. To overcome this limitation, it had been hoped to estimate the influence of changes in the phytoplankton population through the use of measurements of the chlorophyll level in the water column. Figure 5 presents a log-log plot of the zooplankton biomass versus the chlorophyll-a concentration in the water column. These parameters appear to be independent variables. A similar plot (not shown) of the chlorophyll-a concentration versus the concentration of CHC's in the water column on zooplankton yielded a very similar result. If the phytoplankton have a direct role in the pathway of CHC's into the zooplankton community, the chlorophyll-a data shown here do not indicate it.

Whatever the details of the pathway for CHC's from the suspended sediment reservoir into the zooplankton, it seems that only a very small percentage of the CHC's in the water column are associated with the zooplankton (average of 12 ppt PCB in the water column on suspend-

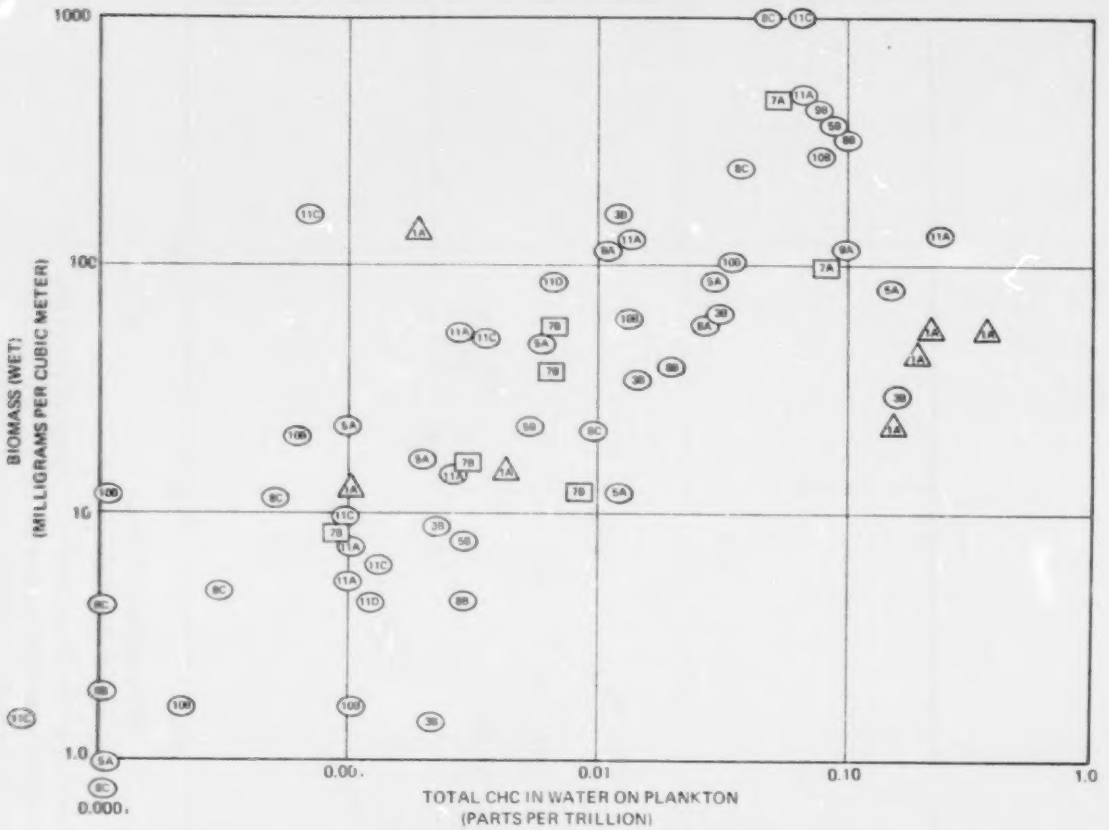

Figure 4. A plot of the log of zooplankton biomass (milligrams weight per cubic meter) versus the log of the total CHC in the water associated with the zooplankton (parts per trillion).

ed sediment versus 0.042 on zooplankton). However, to quantify the rate of movement of the CHC's via this pathway one would need information about turnover rates in the plankton community. Regarding resource management, one should consider that a change in bay conditions that would increase the zooplankton population would probably have the effect of increasing the flow of CHC's from the suspended sediment reservoir into the biological system. This could create adverse consequences as all aquatic species of commercial interest at some time in their lifecycle make up a part of the zooplankton community.

By far, the largest route for the transport of CHC's from the suspended sediment reservoir probably is a result of sediment deposition. The areas of deposition of fine-grain sediments can be visualized as sinks where the CHC's attached to suspended sediments collect. As in the Chester River Study (ref. 1), the chlorinated hydrocarbons in the bottom sediments were found highest where the median grain-size diameters were the lowest. Baltimore harbor is a trap for fine-grain sediments in the upper Chesapeake Bay and, as such, functions as a sink for CHC's from the suspended sediment reservoir. Figure 6 shows that PCB, for instance, was much higher in the sediments of Baltimore harbor than elsewhere in the bay. It would be necessary to analyze core slices and establish the three-dimentional concentration gradient in order to estimate the mass of CHC's contained in the bottom sediment sinks. (A core from the Chester River was found to contain layers of widely varying levels of CHC's to a depth of 50 centimeters—reference 1.)

A number of mechanisms exist for movement of

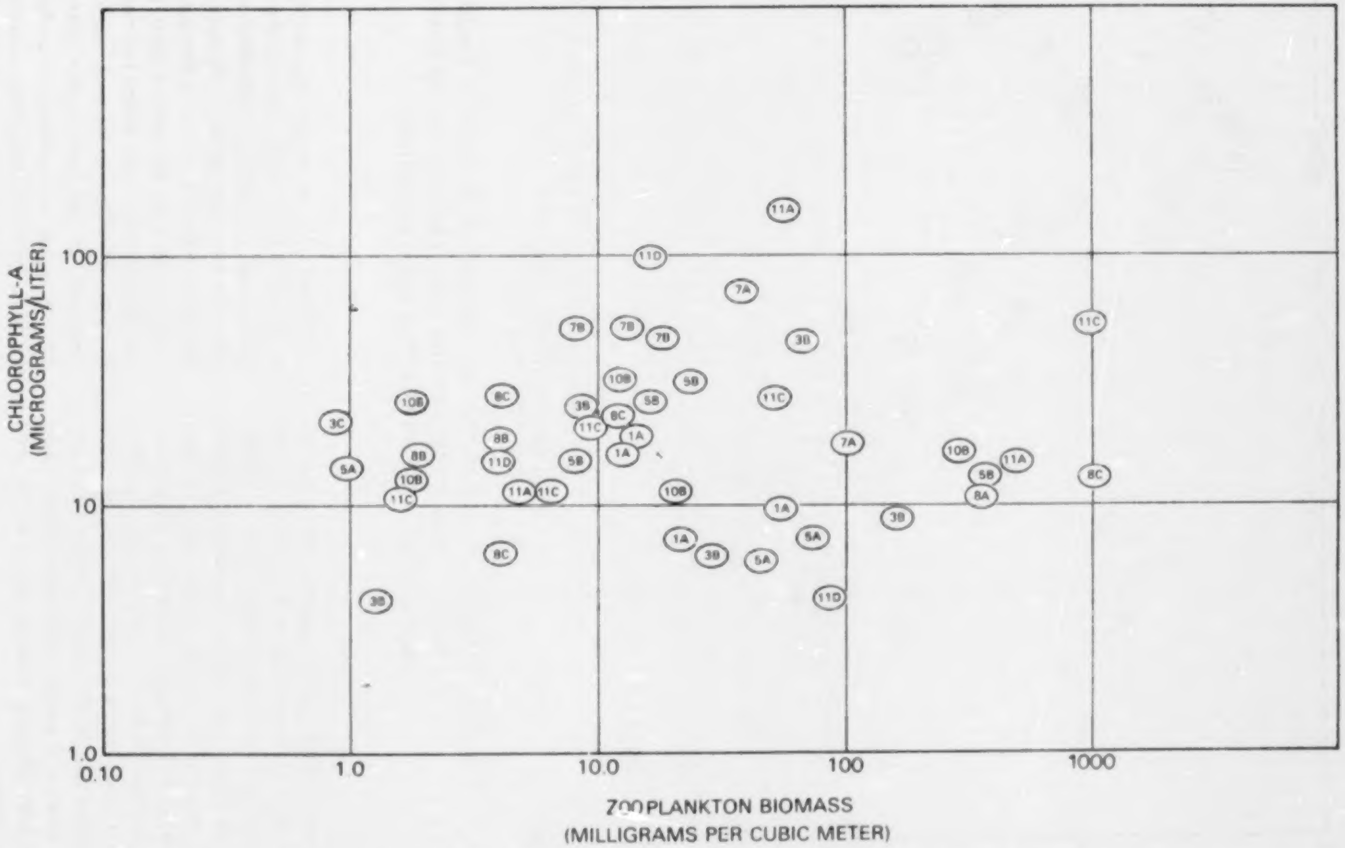

Figure 5. A plot of the log of the chlorophyll–a concentration in the water (micrograms per liter) versus the log of the zooplankton biomass in the water (milligrams per cubic meter).

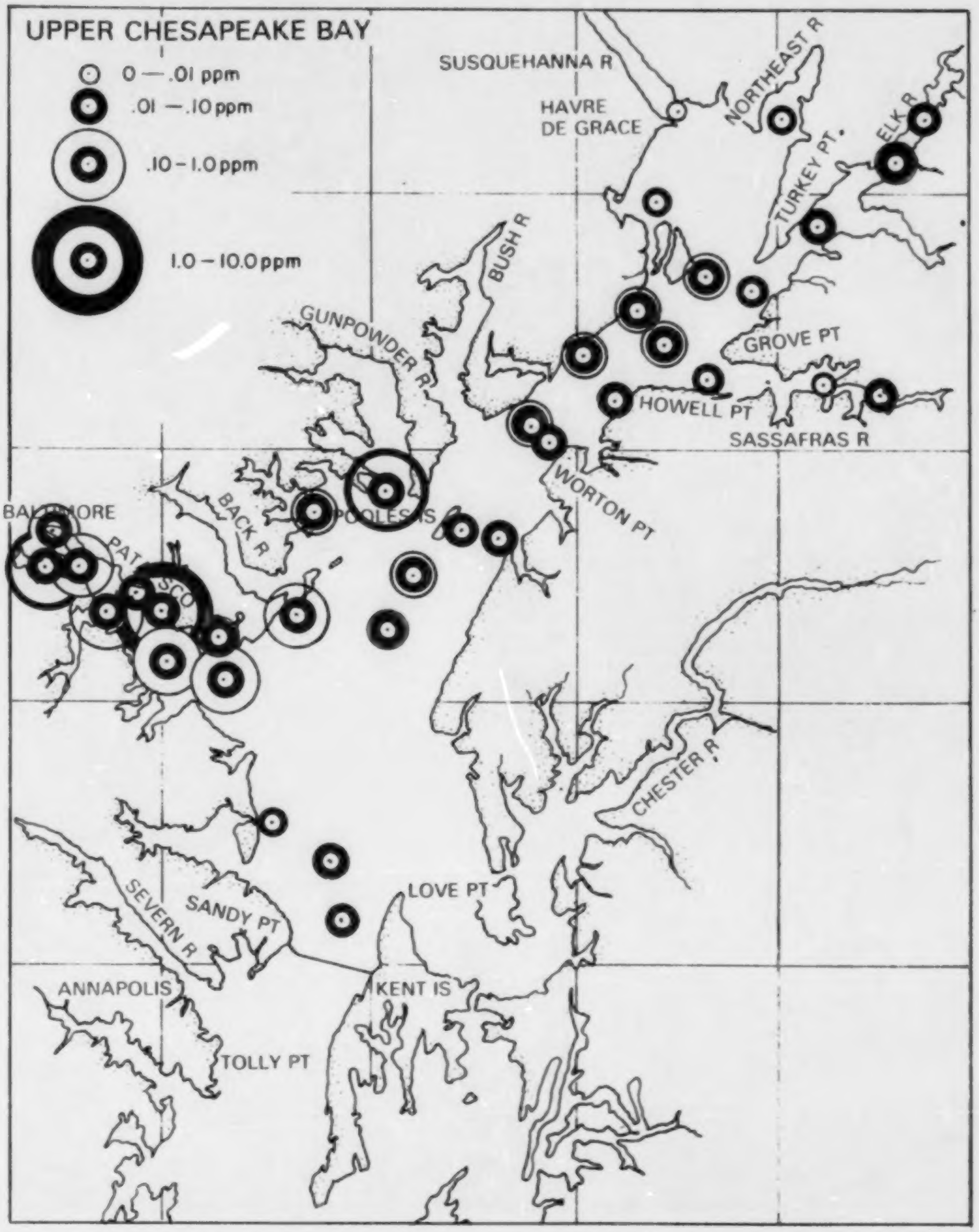

Figure 6. A representation of the concentration of PCB's in bottom sediments samples from the upper Chesapeake Bay.

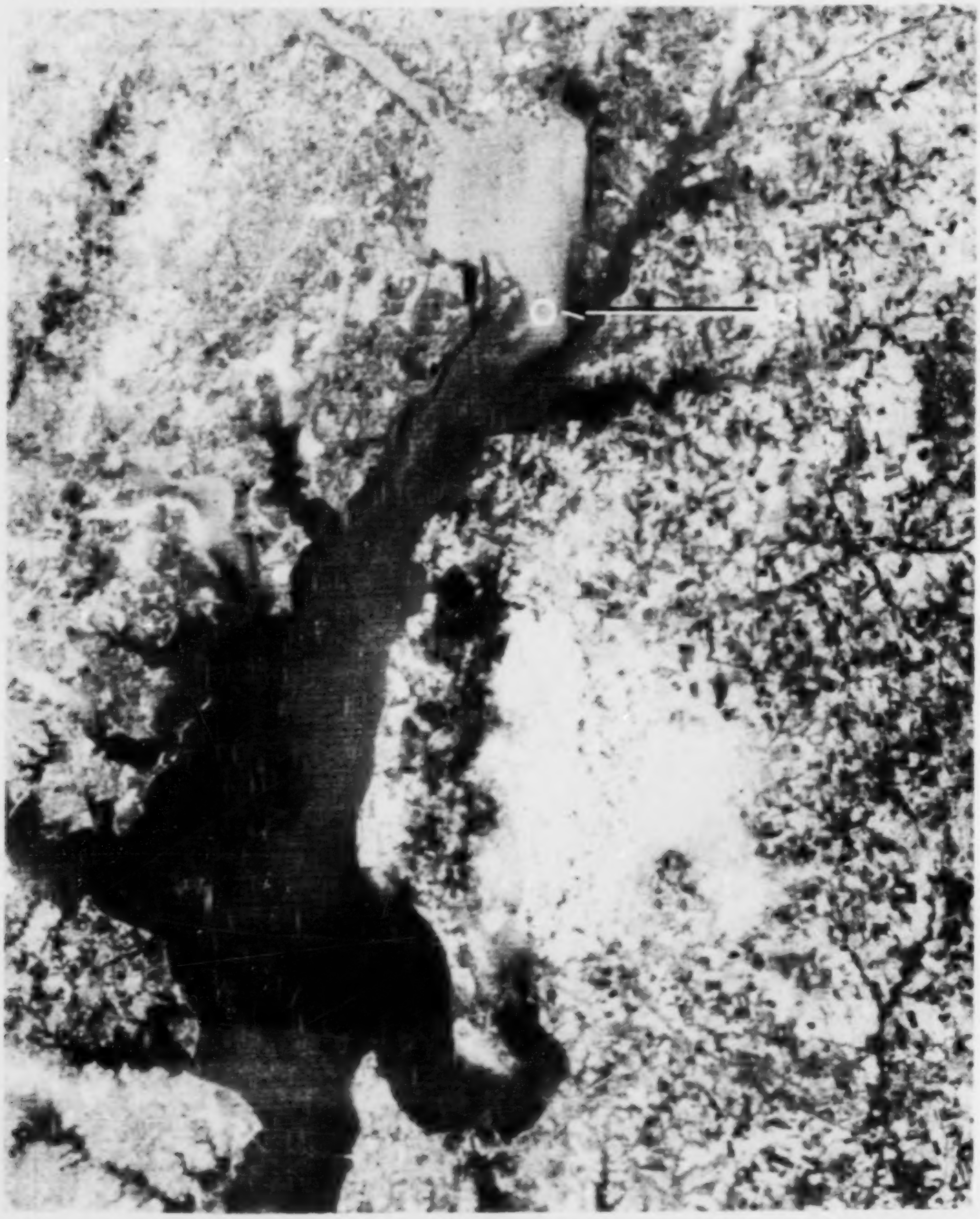

Figure 7. A portion of an Earth Resources Technology Satellite—1 photograph taken April 3, 1974, showing suspended sediment influx into the upper Chesapeake Bay from the Susquehana River. Samples were taken at station "13".

CHC's out of the bottom sediment sinks. When one considers the fine-grain fractions in the upper Chesapeake Bay as a whole, resuspension of this material by tidal scour probably is the major reentry route from the sinks to the suspended sediment reservoir (ref. 7). In the Baltimore harbor area, however, maintenance dredging of navigational channels is a major mechanism for removal of sediments containing chlorinated hydrocarbons from the bottom sediment sink. Although the subsequent fate of this dredged material is a subject clouded by great controversy, during the dredging and subsequent overboard discharge of this material, undoubtedly, a substantial quantity returns to the suspended sediment reservoir.

The Susquehanna River appears to be the major source of chlorinated hydrocarbons to the upper Chesapeake Bay. Nearly all of the suspended sediment in the upper bay originates from the Susquehanna River—entering primarily during the few brief periods of high flow (ref. 7). Figure 7 is an ERTS-1 (Earth Resource Technology Satelite-1) photograph showing the sediment influx at the beginning of a freshet period in April 1974. During this freshet period, suspended sediment samples were collected at the location designed "13" in figure 7. Analysis of these samples (together with other samples collected during the study) indicated that, when they enter the bay, the suspended sediments appear to carry sufficient burden of PCB's and DDT residues to account for levels observed at the lower stations without postulating additional sources. The chlordane values appear low, however, suggesting substantial inputs from other sources. As is discussed in greater detail elsewhere (ref. 4), the urban-industrial activities concentrated in and around Baltimore harbor appear to generate locally high concentrations of PCB and DDT residues, but seem not to contribute a major portion of the total budget of these materials in the upper bay. It seems likely, however, that the harbor area does contribute a large portion of the chlordane found in the upper bay.

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RECENT STUDIES OF TRANSPORT OF PCB'S TO MARINE ENVIRONMENTS

Robert W. Risebrough*

Abstract

The deep ocean waters and sediments are a potential sink for chlorinated biphenyl compounds, removing them from food webs which provide input to man. Data on PCB levels in seawater are as yet unsatisfactory or too few to permit a reliable estimate of the amounts of PCB that have so far entered the oceans. Formulation of a global mass balance equation for the major kinds of PCB compounds would be useful in predicting future contamination levels.

Chlorinated biphenyl compounds have been reported in many marine environments, including those of the Arctic (ref. 1) and the Antarctic (ref. 2); with the DDT compounds they have become contaminants throughout the global ecosystem. Concentrations reported are occasionally high, including those in seawater of the North Atlantic (ref. 3), in marine birds of coastal areas (refs. 4,5,6), in birds feeding on the eggs of other sea birds (ref. 7) and in peregrine falcons (*Falco peregrinus*) of Amchitka Island in the Aleutians (ref. 8).

The high levels which have been reported from both coastal and marine environments raise two questions. Do these levels represent a threat to local ecosystems and to human consumers? And do the high levels indicate that the oceans, particularly the deep waters and marine sediments, can be considered an ultimate sink in which polychlorinated biphenyls are unlikely to enter food webs that provide nourishment to man? Deposition in marine sediments might then be a significant pathway of removal of these pollutants from the biosphere. Attempts to answer the latter question would require formulation of a global mass balance equation of the chlorobiphenyls, which would express the present burden of PCB's in the oceans in terms of global production figures and use patterns. A preliminary formuation of such a global mass balance equation based on North American production and use of PCB has been presented by Nisbet and Sarofim (ref. 9). The present paper reviews some of the work relating to the transport of polychlorobiphenyl compounds to marine environments that has been carried out since Nisbet and Sarofim presented their preliminary model.

Just as it has become misleading to speak of "DDT" in the environment without reference to the individual compounds, so has it become misleading to speak of "PCB" in the environment without reference to the kind of PCB. Thus, the majority of the chlorinated biphenyls used in the United States has consisted of the trichlorinated preparations (ref. 10), whereas PCB reported in marine environments usually consists of penta- or hexachlorobiphenyls. It is therefore meaningless to discuss PCB concentrations in seawater in reference to input from rivers if the former consist predominately of pentachlorobiphenyls and the latter of trichlorobiphenyls.

Nature of PCB Compounds in Marine Environments

The higher vapor pressures of the lower chlorinated PCB compounds (ref. 11) and the production and use figures (refs. 9, 10) suggest that larger quantities of these have entered the marine environment than have the penta- and hexachlorobiphenyls. Thus Bidleman and Olney (ref. 12) found that the majority of the PCB residues in air over the Sargasso Sea consisted of tri- and tetrachlorobiphenyls. Similarly, PCB entering the sea as a component of the wastewaters of Southern California has consisted predominately of trichlorobiphenyls (D. R. Young, R. W. Risebrough, B. W. de Lappe, T. C. Heesen, and D. J. McDermott, unpublished data). Trichlorobiphenyls have occasionally been detected in seawater of the Southern California Bight (ref. 13), but PCB usually reported in seawater has consisted of pentachlorobiphenyls (refs. 3, 14, 15, 16). Bidleman and Olney (ref. 12) have reported that the chlorobiphenyls in their seawater extracts from the Sargasso Sea were predominately hexachlorobiphenyls. Tri- and tetrachlorobiphenyls have been detected in marine fish from the Aleutians and in ptarmigan (*Lagopus mutus*), an arctic grouse that feeds on the tundra (ref. 8). Many extracts of marine species, however, do not contain tri- or tetrachlorobiphenyls in concentrations equivalent or approaching those of the penta- and hexachlorobiphenyls. Bacterial degradation of the lower chlorinated compounds (ref. 17) is a possible pathway of breakdown but the low concentrations of PCB in seawater might not prompt bacteria to synthesize the necessary enzymes for their metabolism, unlike the bacterial response in artificial environments where PCB compounds are the only carbon source. A major difficulty in recording the lower chlorinated compounds in seawater has been the presence in the majority of extracts of high concentrations of other compounds which interfere with the analysis. The higher vapor pressures of the lower chlorinated compounds and their

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longer residence time in the atmosphere might result in higher rates of breakdown by ultraviolet light, but evidence to support this hypothesis is apparently lacking. In higher vertebrates, metabolism to hydroxylated derivatives of unknown significance in the marine environment accounts for the disappearance of some of the tri- and tetrachlorobiphenyls. Formulation of a global mass balance equation of "PCB" therefore makes little sense unless it is done with reference to individual compounds or at least to the major classes of chlorobiphenyls.

Chlorinated Dibenzofurans in the Environment

Other papers presented at this symposium have expressed the current concern over the possible exposure of man to chlorinated dibenzofurans. Preparations of PCB have been shown to produce birth defects in birds (refs. 18, 19, 20), similar to those which have occasionally been observed in populations of terns, *Sterna hirundo*, and *S. dougallii*, breeding in Long Island Sound and feeding on small fish (ref. 21). The observed defects are comparable to those produced by the chlorinated dibenzodioxins (ref. 22). The presence of the chemically related chlorinated dibenzofurans in PCB preparations (refs. 23, 24, 25) suggests that the defects may be caused by these contaminants rather than by chlorobiphenyls.

Extracts of mallards (*Anas platrhynchos*) that had been fed PCB of known dibenzofuran content were examined for the presence of chlorinated dibenzofurans. The chlorinated dibenzofurans originally present in the Aroclor 1254 were found to be metabolized or otherwise excreted at a much faster rate than the pentachlorobiphenyls and were not found in the extracts (ref. 26). It would therefore appear unlikely that these compounds would be accumulated in marine food webs. In the light of other results that have been presented at this symposium, it might be worthwhile to look in the environment for those individual PCB compounds from which dibenzofurans might be formed in vivo, and to examine tissues of species exposed to these compounds for the presence of the furans.

Comparative PCB Levels in Marine Environments

Relative contamination levels of different oceanic areas by PCB compounds have recently been reviewed by Ohlendorf et al. (ref. 27) and by Risebrough et al. (ref. 13); marine birds were used as indicator organisms. In the North Atlantic, PCB contamination appears to increase from west to east; contamination levels in the vicinity of Iceland appear comparable to those in the Aleutians in the North Pacific. In the southern hemisphere, levels in biocoenetic equivalents of northern species are approximately one order of magnitude lower. The difference between the two hemispheres reflects therefore the relative industrialization of the two areas. Data that would permit a more detailed comparison between the Atlantic and the Pacific are as yet insufficient.

PCB in Seawater

Determinations of the PCB levels in seawater have been reported from both the Pacific and the Atlantic. Williams and Robertson (ref. 15) have reported PCB, p,p′-DDT, and p,p′-DDE values in subsurface water from the outer California Current and at two stations in the North Central Pacific Gyre, both in the vicinity of 31° N.155° W. Water was collected in 2.5- liter glass bottles, 10-15 cm below the surface. Reported PCB values in the subsurface water, including filtrate and filtered fractions, were 2.5×10^{-12} g/g in the California Current and 4.5×10^{-12} g/g at one of the North Central Pacific Gyre stations.

Scura and McClure (ref. 16) utilized a column containing 5% activated carbon powder, 10% MgO, and 85% refined diatomaceous earth for the determination of chlorinated hydrocarbons in 1-liter samples of seawater collected off the Southern California coast. Reported PCB concentrations at five stations in the Southern California Bight ranged from 3 ppt to 9.6 ppt (10^{-12} g/g) at the surface; concentrations reported in five samples obtained at depths from 500 to 1,500 meters ranged from 2.3 to 10.3 ppt. In the majority of samples, p,p′-DDE and p,p′-DDT were detected; DDE concentrations ranged from <0.1 ppt to 1.0 ppt at the surface and from 0.3 to 1.2 ppt at depths from 500 to 1,500 meters. Concentrations of p,p′-DDT were somewhat lower.

Bidleman and Olney (ref. 12) have reported PCB and DDT concentrations at eight stations in the Sargasso Sea in 1973. PCB concentrations in subsurface water were lower than detectability ($<0.9 \times 10^{-12}$ g/g) at four of the stations and ranged from 1.0 to 3.6×10^{-12} g/g at the others. Concentrations of p,p′-DDT were 0.5×10^{-12} g/g at one station but were below detectability ($<0.15 \times 10^{-12}$ g/g) at the others. Samples were collected in 3.8-liter glass jugs at depths approximately 30 cm below the surface. Substantially higher concentrations were measured in the surface microlayer. These concentrations were calculated by reference to Aroclor 1260. Concentrations calculated by reference to Aroclor 1254 may have been up to two times higher (ref. 28).

Because of the difficulties in extracting large volumes of seawater with organic solvents, Harvey et al. (ref. 3) developed a column extraction system using Amberlite XAD-2 resin. Seawater samples in volumes ranging between 19 and 80 liters were obtained in a large volume sampler and passed through the resin on board ship. Also, the extracts were analyzed on board ship. Reported PCB concentrations, calculated by reference to Aroclor 1254, averaged 35×10^{-12} g/g at the surface and 10 ppt at 200 meters. At depths of 100, 1,500, and 3,000 meters reported concentrations were $\geqslant 1$ ppt. Samples were obtained in the summer of 1972 (ref. 3).

In 1973 and 1974, substantially lower values were recorded and it was concluded that PCB concentrations in North Atlantic surface waters had declined 40-fold over the 2-year period as a result of restrictions on PCB use (ref. 14). At two stations sampled in 1973 (09° N. 40° W. and 32° N. 70° W.) samples were obtained through the water column. PCB concentrations ranged from 4.3 ppt at 10 meters to 1 ppt at 3,000 meters at the former site and from 0.4 to 1.9 ppt at depths between 10 meters and 5,100 meters at the latter.

Harvey et al. (ref. 14) estimated that the decline of PCB concentrations would require removal of 2×10^4 tons of PCB from the upper 200 meters over the 2-year period, assuming a mean concentration in 1972 of 20×10^{-12} g/g in the upper 200 meters of the North Atlantic between 26° N. and 63° N.; the volume was assumed to be 10^{18} liters. The estimate of the volume over this area at a depth of 200 meters appears, however, to be low, perhaps by an order of magnitude since the corresponding area would be 5×10^6 km^2. The area of the Atlantic, including the North and South Atlantic, is 82×10^6 km^2 (ref. 29). Thus, since the PCB reported consisted predominantly of pentachlorobiphenyls, in the order of 10^5 metric tons of pentachlorobiphenyls would have to be removed from the upper 200 meters. From 1957 through 1974, a total of only 124 million pounds, equivalent to 5.6×10^4 metric tons of pentachlorobiphenyls (Aroclor 1254), was sold in the United States (ref. 10). Sales in 1970, the peak year of U.S. production, amounted to 5.6×10^3 tons. Since the mean depth of the Atlantic is 3,926 meters (ref. 29) and since PCB was recorded at depths to 3,000 meters, the estimated pentachlorobiphenyl content of the North Atlantic would be correspondingly higher. If the average concentration throughout the water column were assumed to be 1 ppt, the average depth assumed to be 3,900 meters and the area of the North Atlantic between the equator and 65° N. were assumed to be in the order of 47×10^6 km^2, the pentachlorobiphenyl content would be in the order of 180,000 metric tons, higher than the total U.S. domestic use.

Similarly, the PCB concentrations reported from the Pacific by Scura and McClure (ref. 16) and by Williams and Robertson (ref. 15) appear to be too high. If a mean value of 1 ppt were assumed for the upper 200 meters of the North Pacific and the area from the equator to 65° N. is 82×10^6 km^2, 16,000 tons of pentachlorobiphenyls would be present in this volume. This represents three times the U.S. use in 1970.

Nisbet and Sarofim (ref. 9) and the Panel on Hazardous Trace Substances (ref. 30) estimated that $1.5\text{-}2.5 \times 10^3$ tons per year of PCB had been discharged into the atmosphere from North America. A large fraction of this amount can be expected to consist of lower chlorinated PCB compounds such as the tri- and tetrachlorobiphenyls.

To eliminate the very significant problems of contamination during collection and extraction, and the difficulties of working with low volumes of seawater, we have worked towards the development of an in situ sampling system, such that all materials in contact with seawater that is sampled can be cleaned in the laboratory to a desired background level and wrapped in clean containers, to be unwrapped only immediately prior to use. The system would extract water at a desired depth and could be rewrapped immediately upon retrieval and stored under appropriate conditions until analysis.

Our own values obtained with this system have been substantially lower than those recorded by other investigators. Thus 23 extracts of seawater obtained off Western Mexico in 1975 contained no detectable chlorobiphenyls; volumes extracted were in the order of 100 liters. In 22 of these samples, concentrations of pentachlorobiphenyls were less than 100 parts per quadrillion (10^{-15} g/g); in 11 samples, concentrations were less than 50×10^{-15} g/g (ref. 13). We believe these values to be more consistent with the requirements of a global mass balance equation, yet intercalibration of all methodologies used to determine organochlorines in seawater is urgently required.

River Transport of Chlorobiphenyls to the Marine Environment

Nisbet and Sarofim (ref. 9) estimated that 4 to 5×10^3 tons/year of PCB were discharged into fresh and coastal waters in the peak year of PCB use. This estimate was based upon use figures and industrial and disposal practices in effect at that time. Of the total PCB, about 1,000 tons may be assumed to have consisted of pentachlorobiphenyls. Analysis of wastewaters and of surface runoff entering the Southern California Bight since 1971 has shown that trichlorobiphenyls have constituted the majority of the total PCB residues. In this area, PCB input into the sea from wastewaters is considerably higher than the input from surface runoff. Estimated total PCB input into the Bight from these sources in 1972 was 20 tons; in 1974 total input was estimated to be 6.5 tons (Young et al., op. cit.).

Atmospheric Transport of PCB's to Marine Environments

Peel (ref. 31) was unable to detect PCB compounds in samples of Antarctic snow obtained in December 1969 inland from the British Antarctic Station at Halley Bay (75°31'S. 26°42'W.). The samples represented the previous 5 to 10 years' accumulation. Total concentrations of p,p'-DDT and p,p'-DDE ranged from 0.1 to 2.0 x 10^{-12} g/g. On this basis, the argument was advanced that on a global scale the atmosphere is not the predominant mode of transport of PCB. These results, therefore, appear inconsistent with those of Bidleman and Olney (ref. 12) and Harvey and Steinhauer (ref. 32), who were able to detect PCB in samples of marine air at concentrations considerably in excess of those of the DDT compounds.

In 1975 we extracted Antarctic snow in situ on Doumer Island in the Antarctic Peninsula (63°35.5'W. 64°51.3'S.); eggs of three species of antarctic penguins (*Pygoscelis adeliae, P. papua, P. antarctica*) were obtained to look for PCB in the food webs and to compare residues with those obtained 5 years previously (ref. 2). Eleven 99-liter samples of snow were extracted by techniques described elsewhere (R. W. Risebrough, W. Walker II, B. W. de Lappe, unpublished manuscript). PCB concentrations, as pentachlorobiphenyls, ranged from 30 to 1,200 x 10^{-15} g/g; median concentration was 300 x 10^{-15} g/g. The median total DDT:PCB ratio in these samples was 6.

Pentachlorobiphenyls were detected in all of 27 penguin eggs of the three species obtained in 1975. In addition, 15 eggs of the Adelie penguin obtained in Arthur Harbor on Anvers Island, near Palmer Station, on 25 December 1973 also contained pentachlorobiphenyls. The median DDE:PCB ratio was 3.0. Concentrations of p,p'-DDT were low compared to those of p,p'-DDE. Furthermore, reexamination of extracts of 14 Adelie penguin eggs obtained at the same site in Arthur Harbor in 1970 showed the presence of PCB compounds in all extracts. A variety of unknown compounds had previously interfered with the detection of pentachlorobiphenyls. These however, could be destroyed by rigorous saponification (R. W. Risebrough and T. T. Schmidt, unpublished data).

Total DDT:PCB ratios recorded in several of the subantarctic samples from the Auckland Islands ranged between 2 and 4 (ref. 33), comparable to those recorded in the Antarctic penguin eggs. Moreover, concentrations of DDE and PCB were equivalent north and south of the Convergence. Thus, fallout patterns of chlorinated hydrocarbons appear to be equivalent north and south of the Antarctic Convergence, which separates waters derived both from the melting of the Antarctic Icecap and from upwelling from waters of the southern Atlantic, Pacific, and Indian Oceans. Residues in the Antarctic have therefore derived from atmospheric transport rather than from oceanic circulation. The possibility that the residues of organochlorine pollutants in Antarctica have resulted from human activities at the scientific bases has been examined and discounted (ref. 34).

Changes in PCB Contamination Levels in the Marine Environment

PCB levels in mussels, *Mytilus*, at several sites in the Southern California Bight, have declined between 1971 and 1974 (B. W. de Lappe, R. W. Risebrough, P. Millikin, and D. R. Young, unpublished data). As discussed above, the reported decline of PCB values in the North Atlantic mixed layer (ref. 14) appears unlikely. The seabird data (refs. 35, 7, 27) do not indicate a decline in the North Atlantic between 1971 and 1973.

Most meetings, particularly international meetings discussing pollution problems of the marine environment, have recommended monitoring programs that would follow with time changes in the levels of particular pollutant classes. These recommendations have yet to be implemented; indeed, we might be reluctant to support such recommendations without assurance that the numbers obtained would be meaningful. A "Mussel Watch" (ref. 36) will shortly be underway in the United States which will examine mussels, principally *M. edulis* and *M. californianus*, from coastal sites for PCB and other chlorinated hydrocarbons, as well as for plutonium isotopes, petroleum compounds, and selected metals. Many variables, including salinity, temperature, particulate content of the water, gonadal condition, time of year, size of the organism, microhabitat, etc., can be expected to affect the variance of the determinations obtained in such monitoring programs. Nevertheless, the program does provide a beginning, which hopefully will determine how levels of chlorobiphenyls change in a marine environment in response to changes in production and use practices.

Summary

The deep ocean waters and sediments are a potential sink for chlorinated biphenyl compounds, removing them from food webs which provide input to man. Data on PCB levels in seawater are as yet unsatisfactory or too few to permit a reliable estimate of the amounts of PCB that have so far entered the oceans. Formulation of a global mass balance equation for the major kinds of PCB compounds would be useful in predicting future contamination levels.

ACKNOWLEDGMENTS

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UPTAKE OF THREE POLYCHLORINATED BIPHENYLS, DDT AND DDE BY THE GREEN SUNFISH, *Lepomis Cyanellus* Raf

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Abstract

The ubiquitous occurrence of polychlorinated biphenyls (PCB's) in aquatic organisms demonstrates the extreme persistence of these materials and their resistance to being metabolized in fish to water-soluble products (refs. 1,2,3,4,5). The complex mixtures of chlorinated biphenyls in commercial PCB's makes quantitative research on the physiological effects of these compounds and their susceptibility to metabolism very difficult. However, the availability of three ^{14}*C biphenyls—2,2′,5-trichlorobiphenyl, 2,5,2′,5′-tetrachlorobiphenyl, and 2,4,5,2′,5′-pentachlorobiphenyl—provides a partial solution to this problem as these three pure isomers are major components of three important commercial PCB's, namely, 1242, 1248, and 1254 (ref. 6). Recently the fate of these pure chlorinated biphenyl isomers has been examined in a model ecosystem. The substantial accumulation of both the tetra- and pentachlorobiphenyl isomers by the mosquito fish,* Gambusia affinis *Baird and Girard, 15.6 and 119.7 ppm (parts per million), respectively, clearly demonstrated the necessity for the examination of the uptake of these materials from water by fish (ref. 7). This communication reports the uptake of three pure* ^{14}*C PCB's, DDT, and DDE by the green sunfish,* Lepomis cyanellus *Raf.*

MATERIALS AND METHODS

The three pure (> 99 percent by tlc, thin-layer chromatography, and radioautography) chlorinated biphenyls were obtained from Mallinckrodt and had the following specific activities in mCi/mM: 2,2′,5-trichlorobiphenyl 9.83; 2,2′,5,5′-tetrachlorobiphenyl 9.78; and 2,4,5,2′,5′-pentachlorobiphenyl 9.78. The ring-labeled ^{14}C DDT and DDE were available from previous work and had specific activities of 0.264 and 5.59 mCi/mM, respectively (ref. 8). Fifteen green sunfish weighing 50-150 mg were placed in 2 liters of aged tap water for a 24-hour acclimation period, then two concentrations, approximately 1 and 3 ppb, of the five compounds in acetone were added to the jars. Each concentration was run at 22 ° C in triplicate and the fish were fed *Daphnia magna* Straus twice a week during the experiment. The experimental design as described in table 1 is taken in part from the study on the uptake of DDT and methoxychlor by several species of fish (ref. 9). This type of experiment models the pulsed introduction of pesticides or industrial chemicals into the aquatic environment. Each point on the graphs represents the average of three fish and when the standard deviation for each point was divided by the average for each point, an average of 22 percent was obtained for all of the experiments.

Analysis of concentrations of the various compounds in the fish was accomplished by solubilizing the fish individually in scintillation vials with one ml Protosol® (New England Nuclear) at 60° C for 4 to 6 hours and then adding Aquasol® (New England Nuclear) for scintillation counting. This method assumes that all the radioactivity is parent compound, which, except for the trichlorobiphenyl which undergoes substantial metabolism, (see table 3) was an excellent assumption. At the end of the experiment the remaining fish were ground in acetone and the extracts were spotted on tlc plates (silica gel GF-254, Brinkmann, 0.25 mm) and developed in Skellysolve B for metabolite distribution by radioautography (Blue Brand X-ray film, Eastman Kodak) and liquid scintillation counting. The fluid used for counting contained PPO (7 g), POPOP (0.05 g), and napthalene (120 g) in 1 liter dioxane. Quenching for both the fish and tlc analysis was corrected using an external standard.

RESULTS AND DISCUSSION

Tables 1 and 2 show the experimental design of the uptake study and the amounts of ^{14}C compounds added to the environment of the fish. The data in table 3 describe the metabolite distribution of the compounds at the end of this uptake investigation. Table 4 contains data showing the bioconcentration of the three ^{14}C PCB's, DDT, and DDE. Figures I through 5 show the time-dependent uptake of the five compounds by *Lepomis cyanellus* Raf. Figure 6 shows the relationship for the three PCB's and DDE between the unextractable radioactivity in *Gambusia affinis* Baird and Girard in the

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Table 1. Timetable for experiment on uptake of three PCB's, DDT, and DDE by *Lepomis cyanellus* Raf

Day	Treatment
0	Add ^{14}C in acetone
2	Take out one fish, weigh and count ^{14}C
3	Take out one fish, weigh and count ^{14}C
9	Change water and add ^{14}C in acetone
10	Take one fish, weigh and count ^{14}C
13	Take one fish, weigh and count ^{14}C
16	Take one fish out, weigh and count ^{14}C, grind remaining fish up in acetone, spot acetone extracts on tlc for metabolite distribution

Table 2. Amounts of three PCB's, DDT, and DDE added to environment of *Lepomis cyanellus* Raf

	First	Second		First	Second
Trichlorobiphenyl	ppb	ppb	DDT	ppb	ppb
Low	0.94	2.80	Low	0.11	0.11
High	2.80	5.61	High	0.33	0.33
Tetrachlorobiphenyl			DDE		
Low	2.50	1.57	Low	1.13	2.25
High	7.50	5.00	High	3.40	6.75
Pentachlorobiphenyl					
Low	1.77	1.77			
High	5.31	5.31			

Table 3. Percent distribution of ^{14}C-labeled PCB's, DDT, and DDE at end of experiment in *Lepomis cyanellus* Raf

	Parent	Polar*
Trichlorobiphenyl	18.39	81.61
Tetrachlorobiphenyl	98.84	1.16
Pentachlorobiphenyl	99.41	0.69
DDE	99.00	1.00
DDT	97.23	2.27

*Rf = 0 Skellysolve B

Table 4. Bioconcentration of ^{14}C-labeled PCB's, DDT, and DDE in *Lepomis cyanellus* Raf. after 15 days

Trichlorobiphenyl	54
Tetrachlorobiphenyl	460
Pentachlorobiphenyl	1,510
DDE	890
DDT	17,500

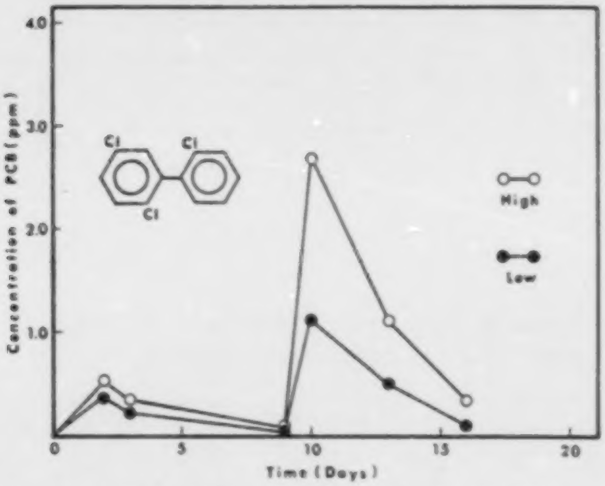

Figure 1. Time-dependent uptake of trichlorobiphency by *Lepomis cyanellus* Raf

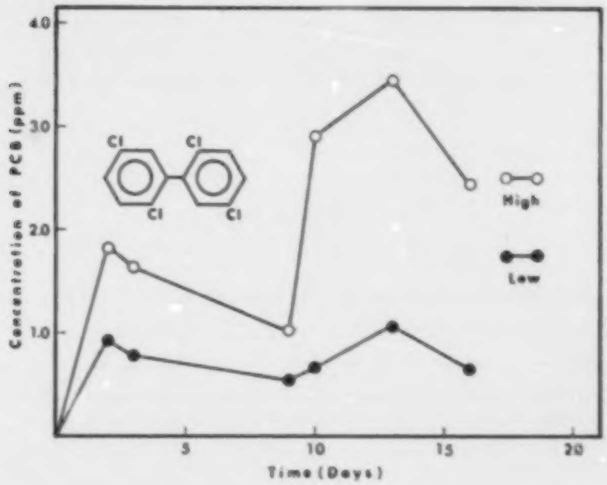

Figure 2. Time-dependent uptake of tetrachlorobiphenyl by *Lepomis cyanellus* Raf

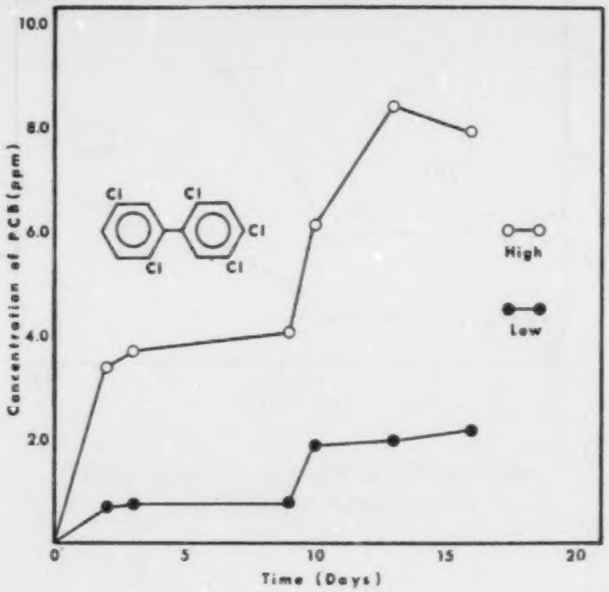

Figure 3. Time-dependent uptake of pentachlorobiphenyl by *Lepomis cyanellus* Raf

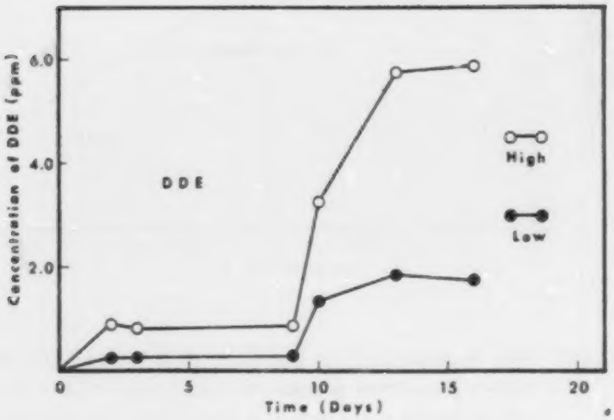

Figure 4. Time-dependent uptake of DDE by *Lepomis cyanellus* Raf

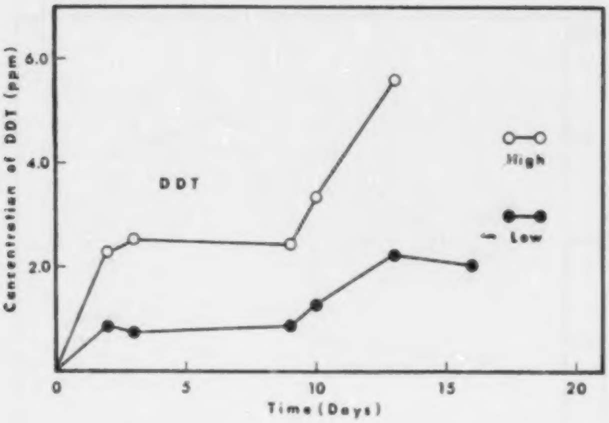

Figure 5. Time-dependent uptake of DDT by *Lepomis cyanellus* Raf

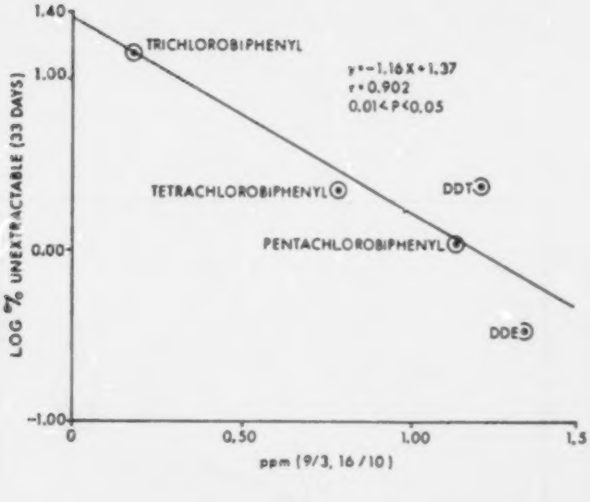

Figure 6. The relationship for the three PCB's and DDE between the unextractable radioactivity in Gambusia affinis Baird and Girard in the 33-day ecosystem and the average change in concentration of radioactivity from day 3 to day 9 and day 10 over day 16.

33-day ecosystem (ref. 7) and the average change in concentration of radioactivity from day 3 to day 9 (ppm day 9/ppm day 3) and day 10 over day 16 (ppm day 16/ppm day 10). Each point on figure 6 on the abscissa is an average of four values.

It is quite clear from examination of figures 1 through 5 that three of the compounds—namely the pentachlorobiphenyl, DDT, and DDE—have similar uptake curves in that the fish retain the radiolabeled compound after it is added to the environment of the fish. The point for the last day for DDT is not shown, as the fish died before final value could be measured. The tetrachlorobiphenyl is retained somewhat less than pentachlorobiphenyl, DDT, and DDE, in that a few days after a peak concentration is reached after addition of labeled biphenyl the curve begins to drop. The trichlorobiphenyl differs dramatically from the preceding four compounds, in that after the peak concentration the curve drops off sharply.

This type of metabolic susceptibility for chlorinated biphenyls, namely that the lower chlorinated biphenyls undergo metabolism more readily than the higher chlorinated PCB's, has been shown for the complex commercial mixtures using rats (ref. 10) and Japanese quail (ref. 11). However, the literature appears to be lacking in metabolism studies on pure isomers of the PCB's except for some work on 4-chlorobiphenyl in the rabbit (refs. 12, 13). A comparative metabolism study with pigeons, rats, and trout and 4-chlorobiphenyl, 4,4′-dichlorobiphenyl, 2,5,2′,5′-tetrachlorobiphenyl, and 2,4,5,2′,4′,5′-hexachlorobiphenyl has been reported (ref. 13). These authors found that the trout could not hydroxylate any of these compounds while the pigeon and rat were able to hydroxylate all but the hexachlorobiphenyl. Recently an elimination study with the rhesus monkey and two pure ^{14}C biphenyls—2,4′-dichlorobiphenyl and 2,5,2′-trichlorobiphenyl—has been reported but no metabolite structure or distribution was reported (ref. 14). The first hydroxylated metabolite from a higher chlorinated biphenyl has been reported in a study of the excretion products of 2,4,3′,4′-tetrachlorobiphenyl in rats (ref. 15). These authors identified two hydroxylated species as the 2- and 5-hydroxy derivatives of the tetrachlorobiphenyl. Further, the 5-hydroxy biphenyl was determined to be about four times more toxic to the rat than the tetrachlorobiphenyl.

The data reported in this paper corroborate quite well the information already available, except for the trout, on the metabolism of PCB's by organisms. In the present work the data indicate rather convincingly that the green sunfish is able to transform the trichlorobiphenyl into polar species as only 18 percent (table 3) of the radioactivity in the fish was parent material at the end of the experiment. Perhaps species differences and temperature optimum of detoxifying enzymes account for the discrepancy between the relative abilities of the trout and green sunfish to metabolize chlorinated biphenyls. Further, previous investigations have shown that trout liver microsomes as compared to mouse liver microsomes are very inefficient at introducing a hydroxyl group into an aromatic nucleus such as biphenyl (ref. 16) and therefore the reported inability of trout to hydroxylate any chlorinated biphenyl is not surprising.

Finally, figure 6 clearly demonstrates an interesting relationship between the dynamics of uptake in the present study, that is, the change in concentration of the biphenyls in the fish after labeled compounds are added to the water of the fish, and the percent unextractable radioactivity in *Gambusia affinis* Baird and Girard in the 33-day model ecosystem experiment. This is a logical relationship, as it would be expected. The greater the metabolic susceptibility, the larger the amount of incorporation of ^{14}C radioactivity into unextractable polar compounds.

This study emphasizes the need for further research and information on uptake and metabolism of polychlorinated biphenyls by aquatic organisms. It has been established that 2,5,2′-trichlorobiphenyl is quite susceptible to metabolism by the green sunfish, and 2,5,2′,5′-tetrachlorobiphenyl and 2,4,5,2′,5′-pentachlorobiphenyl are much less susceptible. These latter two chlorinated biphenyls, especially the pentachloro isomer, resemble the relatively metabolic inert chlorinated hydrocarbons DDT and DDE. Much of the previous work, and this paper, deal with the qualitative assessment of metabolic susceptibility as a function of number of chloro substituents. This type of information is useful as it provides the background data for work that desperately needs to be done on the effect of chlorine isomer distribution on metabolic susceptibility, and, even more important, the further structural elucidation of the hydroxylated chlorinated biphenyls.

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LABORATORY MODEL ECOSYSTEM STUDIES OF THE DEGRADATION AND FATE OF RADIOLABELED TRI-, TETRA-, AND PENTACHLOROBIPHENYL COMPARED WITH DDE

Robert L. Metcalf, James R. Sanborn, Ph.D., Po-Yung Lu, and Donald Nye*

Abstract

Radiolabeled tri-, tetra-, and pentachlorobiphenyls (PCB) and DDE were studied in a laboratory model ecosystem for degradation pathways, and biomagnification in alga, snail, mosquito, and fish. Trichlorobiphenyl was degraded in all the organisms of the model ecosystem much more rapidly than tetrachlor- and pentachlorobiphenyl. Pentachlorobiphenyl was approximately as persistent as DDE. There was a linear relationship between lipid/water partition and ecological magnification and between water solubility and ecological magnification. No evidence of conversion of DDE to PCB was detected.

The laboratory model ecosystem previously described (ref. 1) has been employed for the estimation of the environmental fate of DDT and a number of its analogues (refs. 2,3,4) and for study of aldrin, dieldrin, endrin, mirex, lindane, and hexachlorobenzene (ref. 5). The methodology developed has yielded useful information about (1) the degradation pathways of the various xenobiotics, (2) the toxic effects of the compounds and their degradation products, (3) their comparative biomagnification and food chain concentration, and (4) their comparative biodegradability; all in organisms of five phyla linked in several food chains. This information has proved of value in characterizing the potential environmental pollutant effects of candidate insecticides (refs. 4,6) and of plasticizers (ref. 7). In this paper we report the application of these techniques to a better understanding of the comparative environmental properties of trichloro-, tetrachloro-, and pentachlorobiphenyl (PCB's), and of dichlorodiphenyl-dichloroethylene (DDE) the persistent DDT degradation product.

Methods and materials

The laboratory model ecosystem evaluation was carried out in a small glass aquarium with a sloping terrestrial-aquatic interface of pure white sand exactly as previously described (ref. 1). The ^{14}C-radiolabeled compounds were applied quantitatively from acetone solution at 5.0 mg (or ca. 1 kg per ha) to *Sorghum vulgare* seedlings grown in the terrestrial portion. The treated leaves were consumed by fourth instar salt marsh caterpillar larvae *Estigmene acrea,* whose activities and fecal products contaminated the aquatic portion of the system.

The radiolabeled products were transferred through several food chains, e.g., alga (*Oedogonium cardiacum*) → snail (*Physa*); plankton → water flea (*Daphnia magna*) → mosquito (*Culex pipiens quinquefasciatus*) → fish (*Gambusia affinis*). After 33 days in an environmental chamber at 26° C and a 12-hr photoperiod at 5,000 foot candles simulated daylight, the organisms were extracted with acetonitrile and the ^{14}C-radiolabeled compounds evaluated by TLC on silica gel containing fluorescent marker (E. Merck GF-254) and radioautography on no-screen X-ray film. Liquid scintillation counting of the individual components was done in cocktail D (5 g PPO and 100 g naphthalene in dioxane to make 1 liter) and counts were corrected to dpm by using channels ratio quenching correction. The residues, after extraction, were counted by total combustion to $^{14}CO_2$ by the Schoniger oxygen flask technique (ref. 8) to determine the unextractable radioactivity. Whenever possible, the identity of individual components on the TLC plates was determined by cochromatography with known standards and by extraction and mass spectrometry.

Radiolabeled Compounds. The individual ^{14}C-labeled PCB's were obtained from Mallinkrodt, St. Louis, Missouri. They were: 2, 5,2′-trichlorobiphenyl (2,5-dichlorophenyl-ring-UL-^{14}C), 9.91 mCI per mmole with >98 percent radiopurity and 41.5 percent Cl, and a principal constituent of Aroclor 1242 (ref. 9); 2,5,2′,5′-tetrachlorobiphenyl (ring-UL-^{14}C), 9.87 mCi per mmole with >98 percent radiopurity and 48.7 percent Cl, and a principal constituent of Aroclor 1248 (ref.9); and 2,4,5,2′,5′-pentachlorobiphenyl (2′,5′-dichlorophenyl- ring-UL-^{14}C), 9.87 mCi per mmole with >98 percent radiopurity and 54.4 percent Cl, a principal constituent of Aroclor 1254 (ref. 9).

^{14}C - labeled 2,2-bis-(p-chlorophenyl)-1,1-dichloroethylene (DDE) was prepared from ^{14}C-ring-UL p,p′-DDT obtained from the Radiochemical Centre, Amersham, England, 5.48 mCi per mmole, by dehydrochlorinating with 1.0 M alcoholic KOH, and purifying on

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a silicic acid column with hexane elution to 99 percent radiopurity.

Results

PCB's. The movement of ^{14}C radioactivity from *Sorghum* plants into the water phase of the model ecosystem is shown in figure 1. All three chlorinated biphenyls reached a maximum concentration in water at about 7 days after treatment and the levels of contamination declined as the PCB's were taken up by the organisms of the system. The levels of the chlorinated biphenyls in the water phase (table 1) were in the ppb range, below the water solubility of the compounds as determined by radiotracer technique (table 2).

Radioautographs of the extracts from the components of the model system after TLC are shown in figure 2. The data in table 1 represent the quantitative distribution of the ^{14}C in the spots on the TLC plates. The results for the three PCB's are also expressed in table 2 in terms of ecological magnification (E.M.) (ppm in organism/ppm in water) and of biodegradability index (B.I.) (ppm polar degradation products/ppm nonpolar products). The E.M. values for the parent compounds increased substantially with the number of chlorine atoms, from trichlorobiphenyl (41.5 percent Cl) to tetrachlorobiphenyl (48.7 percent Cl) to pentachlorobiphenyl (54.4 percent Cl). Conversely, the B.I. values decreased with increasing degree of chlorine. This consistent and regular behavior gives added confidence that these parameters are ecologically significant (ref. 4) and must be a function of the number of C-H bonds available for hydroxylation by microsomal oxidations in the various organisms. The spots of low R_f value (0.02-0.06), figure 2, are presumably hydroxylated PCB compounds and the polar radioactivity (R_f 0.0) is thought to consist of conjugates of these compounds. Wallnofer et al. (ref. 10) have found 4-chloro-4′-hydroxybiphenyl as a metabolite of 4-chlorobiphenyl from soil fungus, *Rhizopus japonicus*. Yoshimura and Yammamoto (ref. 11) have reported the 5-hydroxylated derivative as the major and the 3-hydroxylated derivative as the minor excretion product of 2,4,3′,4′-tetrachlorobiphenyl in the rat. Hutzinger et al. (ref. 12) have shown that rat and pigeon could hydroxylate 2,5,2′,5′-tetrachlorobiphenyl but they could not detect hydroxylated metabolites in brook trout. However, the

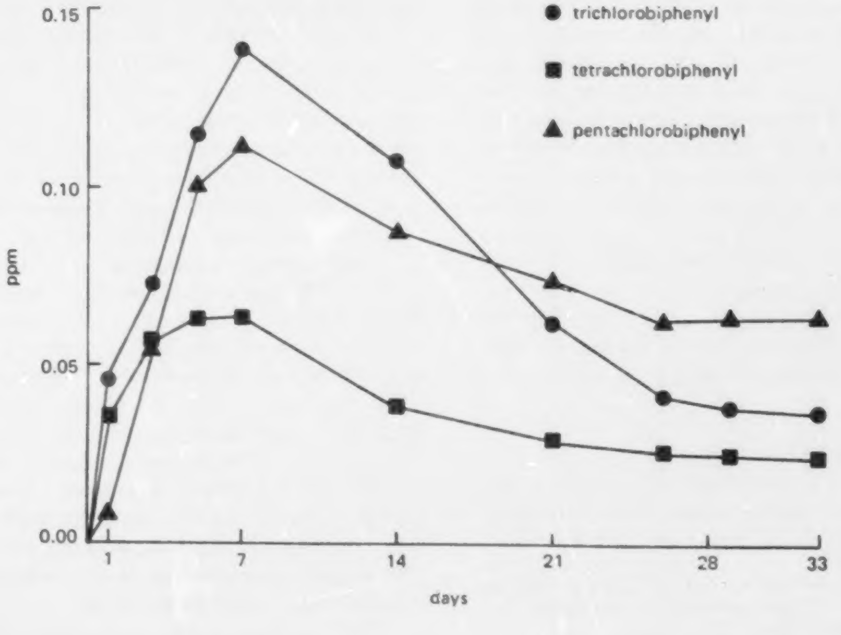

Figure 1. Movement of total ^{14}C radioactivity from plants into the water phase of the model ecosystem and uptake by organisms.

Table 1. Distribution of chlorinated biphenyls and their degradation products in the model ecosystem

	Chlorinated biphenyl equivalents (ppm)				
	H_2O	*Oedogonium* (alga)	*Physa* (snail)	*Culex* (mosquito)	*Gambusia* (fish)
I. 2,5,2′-trichlorobiphenyl total ^{14}C	0.03845	23.2155	31.2015	2.7030	3.2055
Unknown I (R_f 0.66[a])	0.00015	15.9575	18.9720	1.1995	0.2085
trichlorobiphenyl (R_f 0.56)	0.00020	1.4630	1.1590	0.1630	1.2800
Unknown II (R_f 0.23)	0.00005	0.0520	0.6480	+	0.1595
Unknown III (R_f 0.10)	–	–	0.9735	–	–
Unknown IV (R_f 0.06)	0.00055	–	0.5460	–	–
Unknown V (R_f 0.04)	0.00040	–	0.2205	–	–
Unknown VI (R_f 0.03)	0.00040	0.0685	0.4410	–	–
Polar (R_f 0.0)	0.02265	0.5185	3.9315	0.4795	0.9985
Unextractable	0.01405	5.1560	4.3100	0.8610	0.5590
II. 2,5,2′,5′-tetrachlorobiphenyl total ^{14}C	0.02065	23.6845	53.7465	14.5335	15.5685
tetrachlorobiphenyl (R_f 0.48[a])	0.00120	21.5975	47.3275	12.6745	14.2360
Unknown I (R_f 0.23)	0.00005	0.3220	0.7560	0.1070	0.0890
Unknown II (R_f 0.04)	0.00155	0.1030	0.4360	–	–
Polar (R_f 0.0)	0.01225	0.3275	3.9850	0.9670	0.8545
Unextractable	0.00560	1.3345	1.2420	0.7850	0.3900
III. 2,5,2′,4′,5′-pentachlorobiphenyl total ^{14}C	0.04340	62.4660	633.0165	181.4565	127.6945
pentachlorobiphenyl (R_f 0.55[a])	0.00985	53.8440	587.3545	170.8480	119.7060
Unknown I (R_f 0.46)	–	0.6850	8.6210	2.4070	2.5380
Unknown II (R_f 0.39)	0.00020	0.5080	2.2490	1.3195	0.5810
Unknown III (R_f 0.21)	0.00015	0.1425	1.9365	1.0520	0.3285
Unknown IV (R_f 0.04)	0.00030	–	0.5000	–	–
Unknown V (R_f 0.02)	0.00385	0.2570	7.4965	–	0.7450
Polar (R_f 0.0)	0.02055	1.6265	16.5550	2.6745	2.3610
Unextractable	0.00850	5.4330	8.3040	3.1555	1.4350

[a] TLC with hexane (Skellysolve B, bp 60-68°C).

Table 2. Ecological magnification (E.M.) and biodegradability index (B.I.) of PCB's and DDE compared with water solubility and partition coefficient

Chemical	H_2O solubility (ppb)	Partition coefficient	Ecological magnification (E.M.)				Biodegradability index (B.I.)			
			Alga	Snail	Mosquito	Fish	Alga	Snail	Mosquito	Fish
tri-Cl-PCB	16	7,803	7,315	5,795	815	6,400	0.30	0.17	0.35	0.60
tetra-Cl-PCB	16	8,126	17,997	39,439	10,562	11,863	0.015	0.082	0.076	0.060
penta-Cl-PCB	19	16,037	5,464	59,629	17,345	12,152	0.029	0.027	0.0134	0.019
DDE	1.3	18,893	11,251	36,342	59,390	12,037	0.069	0.049	0.033	0.050

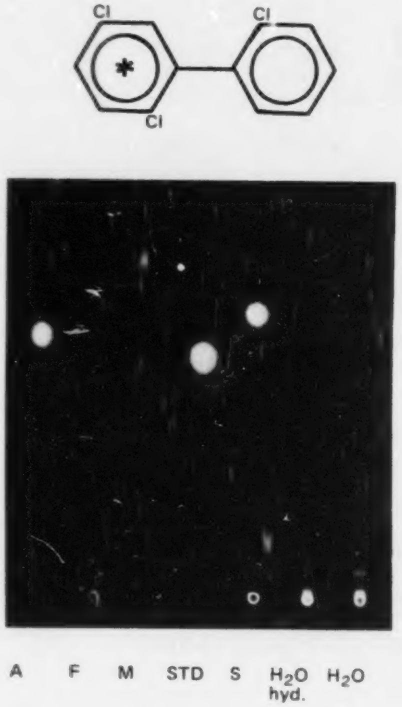

Figure 2A. Radioautogram of TLC plate containing extracts of water and organisms treated with 2,5,2′-trichlorobiphenyl. A (alga), F (fish), M (mosquito larva), S (snail) and STD (^{14}C-radiolabeled compound).

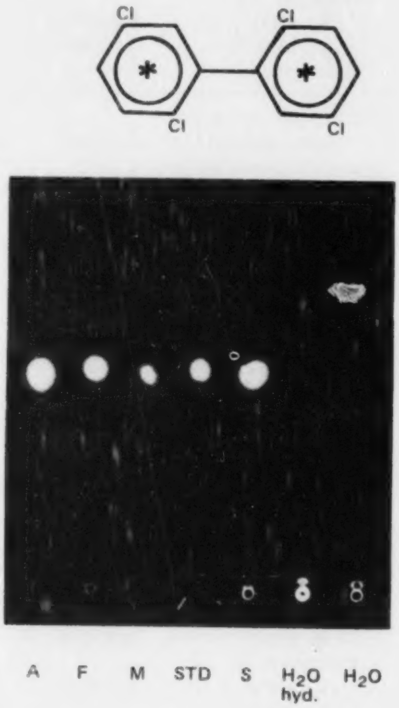

Figure 2B. Radioautogram of TLC plate containing extracts of water and organisms treated with 2,5,2′,5′-tetrachlorobiphenyl. A (alga), F (fish), M (mosquito larva), S (snail) and STD (^{14}C-radiolabeled compound).

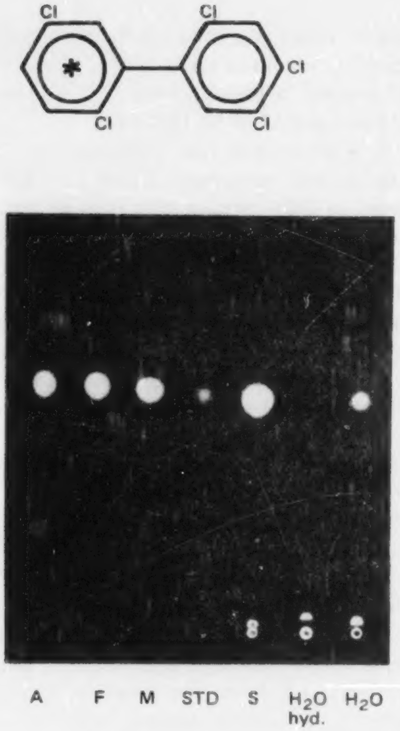

Figure 2C. Radioautogram of TLC plate containing extracts of water and organisms treated with 2,4,5,2',5'-pentachlorobiphenyl. A (alga), F (fish), M (mosquito larva), S (snail) and STD (^{14}C-radiolabeled compound).

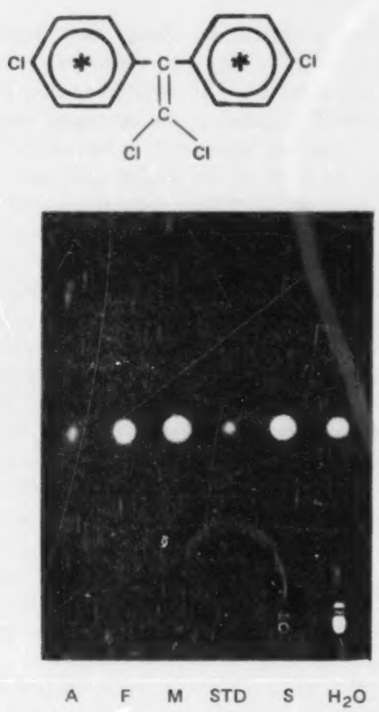

Figure 2D. Radioautogram of TLC plate containing extracts of water and organisms treated with DDE. A (alga), F (fish), M (mosquito larva), S (snail) and STD (^{14}C-radiolabeled compound).

amounts of polar material in *Gambusia* (figure 2, table 1) suggest that this fish is able to slowly hydroxylate this tetrachlorobiphenyl.

The pentachlorobiphenyl with B.I. values of 0.019 to 0.027 in fish and snail is very comparable in model ecosystem behavior to DDT, B.I. 0.015 and 0.044 (ref. 4) and this suggests that the two compounds should behave similarly in the environment (ref. 13). Properties of the tetrachlorobiphenyl were similar to those of the pentachlorobiphenyl (figure 2) but the trichlorobiphenyl was much more degradable. A prominent degradative product (R_f 0.66) is stored in alga, snail, and mosquito larva in much greater quantities than the parent compound. This compound is less polar (higher R_f) in the hexane solvent than any of the three PCB isomers. As shown in table 1, it is magnified to very high values, 106,382X in alga and 126,480X in snail, is stored in lipids, and is highly persistent. Its presence in high amounts in alga and in the snail and mosquito, which are alga feeders, suggests that it might be formed by photochemical processes during photosynthesis in the alga. This compound forms slowly and no traces of it were visible in 3-day uptake studies of trichlorobiphenyl by alga, snail, daphnia, mosquito, or fish (ref. 14) although it appeared in alga in 14-day studies. To date we have been unsuccessful in identifying the unknown by mass spectrometry.

DDE. This compound has been implicated as a possible environmental precursor of PCB isomers through photooxidation reactions involving radical rearrangements to 3,6-dichlorofluorenone intermediates (refs. 15,16,17). Although such rearrangements could logically produce 4,4'-dichlorobiphenyl, it is difficult to see how trichloro- and tetrachlorobiphenyls could be formed as suggested by Maugh (ref. 18). Moreover, Kerner et al. (ref. 19) could detect only bis-(p- chlorophenyl)-chloroethylene (DDMU) after ultraviolet irradiation of DDE. Because of the ecological importance of these possible rearrangements, we have reinvestigated the behavior of DDE in the model ecosystem (ref. 1) to determine if any PCB-like products could be formed under the simulated daylight of the model ecosystem (5,000 foot candles) in an environmental chamber. The radioautograph showing the fate of pure DDE is presented in figure 2. When the extracts of water and organisms were developed on TLC plates with Skellysolve B (hexane fraction) there was no trace of any ^{14}C-labeled compounds with R_f values between 0.05 and 0.47 (DDE) or of any less polar materials with higher R_f values. Under these conditions, as shown in figure 2, trichlorobiphenyl has R_f 0.43, tetrachlorobiphenyl R_f 0.50, and pentachlorobiphenyl R_f 0.53. Detection levels with the techniques used are approximately 0.1 ng (e.g., spot at alga origin in DDE, figure 2) or about 0.00002 percent of the total ^{14}C applied. Thus under the model ecosystem conditions, there is no evidence of formation of PCB isomers from DDE.

DDE is extremely stable in the tissues of the living organisms of the model ecosystem and is stored as approximately 92, 93, 95, and 97 percent of the total ^{14}C in snail, alga, fish, and mosquito larva. The percent of unextractable ^{14}C in these organisms ranged from 0.10 to 0.93 (table 3). The B.I. value for DDE in fish was 0.049 and the E.M. value 12,037 (compared with 0.032 and 27,358 found by Metcalf et al.) (ref. 1). From these values it is apparent that DDE is a more stable environmental pollutant than 2,4,5,2′,5′-pentachlorobiphenyl (table 1), which was stored in the organisms at 86 to 94 percent of the total radioactivity, with from 1.12 to 8.67 percent of unextractable ^{14}C, and had a B.I. of 0.019 and an E.M. of 12,152 in fish.

It is of interest that Sodergren (ref. 20), using a model aquatic ecosystem, found no major metabolic changes in DDE occurring in passage through a food chain into fish, although similar experiments with a polychlorinated biphenyl mixture (Clophen A) showed that the lower fractions with low chlorine content were degraded when transported through the food chain, as was 2,5,2′-trichlorobiphenyl in our experiments. However, in our studies (figure 2, table 3), the water phase contained several polar radiolabeled degradation products. These were resolved on silica gel into at least 11 distinct compounds using a solvent of benzene:dioxane:acetic acid (90:30:1) and we are presently attempting to identify the pathway of DDE degradation in the environment.

Biomass Recovery. To determine the relative availability of the various organisms of the model ecosystem as reservoirs for the bioaccumulation of the micropollutants studied, the total amounts of ^{14}C-labeled products recovered from the principal organisms of the model ecosystems treated with tri-, tetra-, and pentachloro-PCB's, and with DDE were evaluated, as shown in table 4. The evaluations were made on the basis of total recovery of the applied pollutant; of recovery of the maximum amount of pollutant in water (figure 1) for each of the four principal organisms, alga, snail, mosquito, and fish; and of biomass recovery (four organisms) of the total amount of pollutant lost from water (figure 1). The figures of table 4 are very revealing

Table 3. Distribution of DDE and degradation products in the model ecosystem

	DDE equivalents (ppm)				
	H_2O	*Oedogonium* (alga)	*Physa* (snail)	*Culex* (mosquito)	*Gambusia* (fish)
Total ^{14}C	0.00384	7.4720	38.1958	24.8588	7.8653
DDE (R_f 0.49[a])	0.00062	6.9759	22.5325	36.8223	7.4632
Unknown I (R_f 0.05)	0.00009	–	0.8035	–	–
Polar (R_f 0.0)	0.00223	0.4881	1.1612	1.2448	0.3746
Unextractable	0.0009	0.0080	0.3616	0.1087	0.0275

[a] TLC with hexane (Skellysolve B, bp 60-68°C).

Table 4. Biomass recovery of chlorinated biphenyls, and DDE from organisms of model ecosystem

% Recovery	Alga	Snail	Mosquito	Fish
			trichlorobiphenyl	
^{14}C in solution	0.18	0.015	0.0017	0.12
total ^{14}C	0.033	0.0028	0.00032	0.021
(biomass) of ^{14}C lost from solution ———		0.45		
			tetrachlorobiphenyl	
^{14}C in solution	3.33	1.04	0.23	1.91
total ^{14}C	0.28	0.088	0.019	0.16
(biomass) of ^{14}C lost from solution ———		8.7		
			pentachlorobiphenyl	
^{14}C in solution	4.57	19.0	2.32	11.8
total ^{14}C	0.74	3.06	0.37	1.90
(biomass) of ^{14}C lost from solution ———		57.2		
			DDE	
^{14}C in solution	22.4	4.03	2.25	20.2
total ^{14}C	0.24	0.044	0.055	0.22
(biomass) of ^{14}C lost from solution ———		65.8		

in terms of the biodegradability of the various compounds. The highest recoveries of the ^{14}C lost from solution were obtained from the organisms with DDE, 65.8 percent, and pentachlorobiphenyl, 57.2 percent. With tetrachlorobiphenyl, recoveries of 8.7 percent were still substantial, but with trichlorobiphenyl (recovery 0.45 percent), the compound was nearly completely degraded and excreted.

Degradation in Salt Marsh Caterpillar. This animal was chosen, after considerable study, as the dispersing agent for the model ecosystem because it was able to ingest a large variety of organic compounds without apparent injury (ref. 14). The effects of passage of the PCB isomers through the insect are of interest as representing the first stage in the biodegradation of these compounds. Figure 3 shows radioautographs of TLC plates of extracts of feces and body homogenates from larvae feeding on about 30 μg of ^{14}C PCB incorporated in a synthetic diet. Figure 3 and the quantitative evaluation of the radioactivity in the various spots shown in table 5 demonstrate conclusively the much greater degradability of the trichlorobiphenyl over the tetrachlorobiphenyl and pentachlorobiphenyl. With the trichloro compound, the caterpillar feces contained 91 percent of the recovered ^{14}C, with the remainder in the body homogenate, while with the tetrachloro- and pentachlorobiphenyls, the feces contained 21 percent and 24 percent of the radioactivity. The unknown (R_f 0.05) found in feces after trichlorobiphenyl is probably the principal hydroxylated degradation product leading to the very large amount of polar radioactivity. Whereas only low levels of trichlorobiphenyl were retained in the salt marsh caterpillar body, with tetrachloro- and pentachlorobiphenyl the major portion of ^{14}C was retained in the insect body.

DDE passed through the salt marsh caterpillar largely unchanged, with 81 percent of the total radioactivity recovered retained in the body homogenate and 19 percent in the fecal excreta (table 5).

Ecological Magnification. The uptake and concentration of organic compounds by living organisms either directly or through food chains appear to be a function of two important factors: (1) their high lipid solubility and low water solubility, e.g., a large lipid/water partition coefficient, and (2) their resistance to degradation by enzymatic processes, especially the multifunction

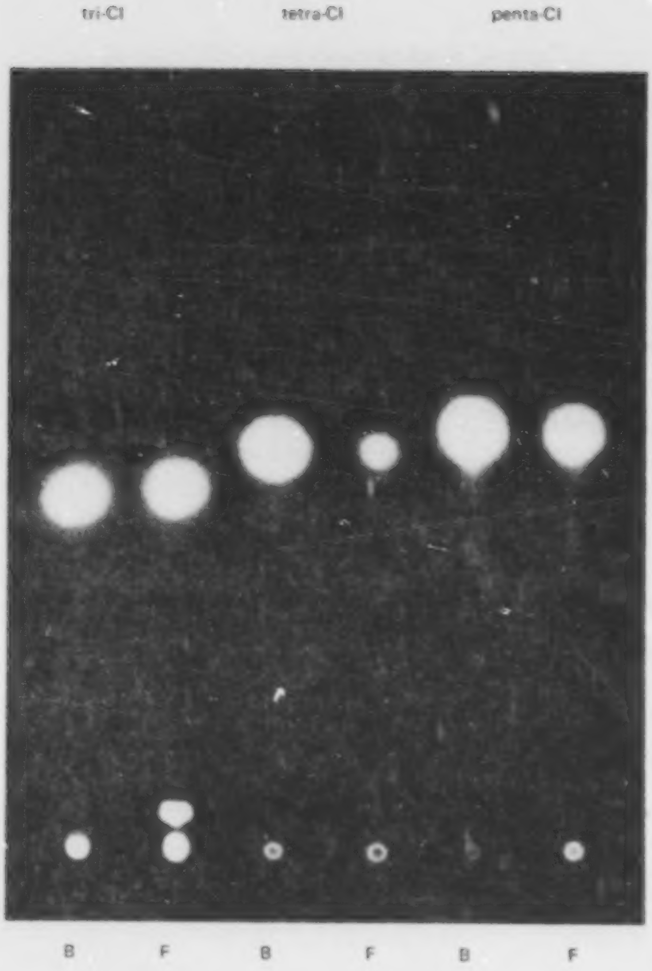

Figure 3. Radioautogram of TLC plate containing extracts of bodies and feces of salt marsh caterpillar larvae fed ^{14}C-labeled 2,5,2′-tri-, 2,5,2′,5′-tetra-, and 2,4,5,2′,5′-pentachlorobiphenyls. B (body homogenate), and F (fecal excreta).

Table 5. Metabolism of ^{14}C-radiolabeled compounds by salt marsh caterpillar[a]

	Body	Feces
A. 2,5,2′-trichlorobiphenyl total ^{14}C (%)	8.66	91.34
Unknown I (R_f 0.53[a])	0.64	–
trichlorobiphenyl (R_f 0.43)	5.84	8.91
Unknown II (R_f 0.31)	0.27	0.37
Unknown III (R_f 0.13)	0.05	0.12
Unknown IV (R_f 0.05)	0.10	4.67
Unknown V (R_f 0.02)	0.11	0.92
Polar (R_f 0.0)	1.65	76.35
B. 2,5,2′,5′-tetrachlorobiphenyl total ^{14}C (%)	78.68	21.32
tetrachlorobiphenyl (R_f 0.50[a])	75.60	15.08
Unknown I (R_f 0.41)	0.99	1.36
Unknown II (R_f 0.05)	0.13	–
Unknown III (R_f 0.03)	trace	0.20
Polar (R_f 0.0)	1.96	4.64
C. 2,5,2′,4′,5′-pentachlorobiphenyl total ^{14}C (%)	75.86	24.14
pentachlorobiphenyl (R_f 0.53[a])	74.00	20.70
Unknown I (R_f 0.46)	0.74	0.74
Unknown II (R_f 0.39)	0.62	0.56
Unknown III (R_f 0.03)	0.08	0.08
Polar (R_f 0.0)	0.42	2.06
D. 2,2-*bis*-(*p*-chlorophenyl)-1,1-dichloroethylene (DDE) total ^{14}C (%)	80.59	19.41
DDE (R_f 0.49[a])	76.88	19.37
Polar (R_f 0.0)	3.71	0.04

[a] TLC with hexane (Skellysolve B, bp 60-68°C).

oxidase enzymes (ref. 14). Hamelink et al. (ref. 21) have suggested that the water insolubility of highly lipid-soluble compounds provides the driving force in producing lipid storage, through a series of simple partitionings from water to lipids. We have correlated the E.M. values for the PCB's and DDE from the fish of the model ecosystems with both water solubility (table 2) in figure 4, and with the octanol/water partition value (table 2) in figure 5. Because the values for the PCB's and DDE fall closely together, the relationships have been extended using values for aniline, anisole, benzoic acid, chlorobenzene, and nitrobenzene taken from other model ecosystem studies (ref. 22). For the limited number of compounds included, the correlation between physical properties and biomagnification is excellent. The regression equation for log water solubility vs. log E.M. (figure 4) was:

$$y = 4.4806 - 0.4732X: \quad n = 9, \quad r = -0.9677.$$

The regression equation for log partition coefficient (Hansch's π) vs. log E.M. (figure 5) was:

$$y = -0.7504 + 1.1587X: \quad n = 9, \quad r = 0.9771.$$

Thus for the organic compounds studied, the properties of water solubility and octanol/water partition coefficient appear to provide a realistic estimate of the biological magnification found in living organisms.

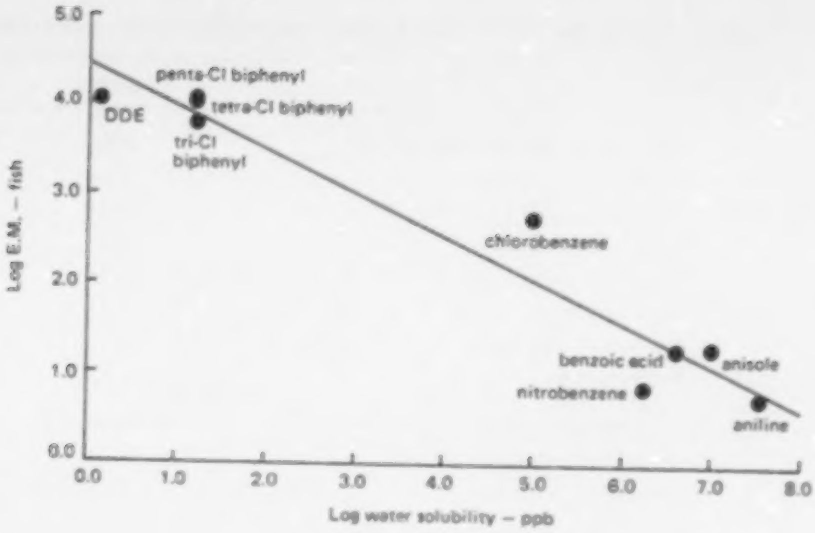

Figure 4. Plot of log E.M. (ecological magnification) for fish vs. log water solubility (ppb).

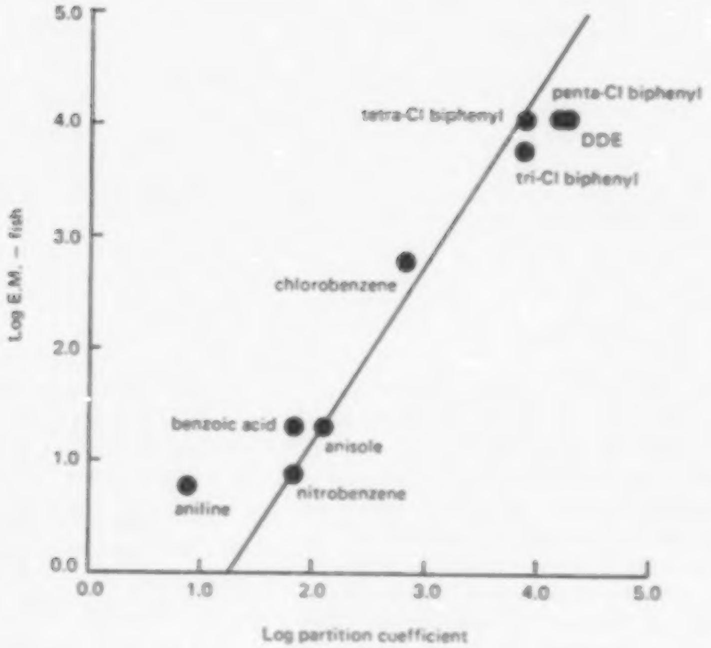

Figure 5. Plot of log E.M. (ecological magnification) for fish vs. log octanol/water partition coefficient.

ACKNOWLEDGMENTS

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ENVIRONMENTAL TRANSPORT AND OCCURRENCE OF PCB's IN 1975

Ian C. T. Nisbet, Ph.D.[*]

Abstract

Despite curtailment of some dispersive uses, environmental levels and rates of transport of PCB's have not changed greatly since 1972. This is probably due in part to the continued release of materials in service prior to 1971 and in part to time lags in environmental transport and dissipation.

Surprisingly high concentrations of PCB's matching Aroclor 1254 are still being found in air, in precipitation, and in dry fallout.

As yet, there is little evidence that significant quantities of PCB's are being leached from dumps.

Human exposure to PCB's is higly variable. Some sport fishermen and breast-fed infants have especially high intakes.

Almost nothing is known about the environmental behavior of chlorinated dibenzofurans.

I think the organizers of the conference may have expected me to compose a global transport model while this session was going on. I have not attempted to do this, except to make some quick calculations on the back of an envelope.

My general conclusion from what I have heard today and yesterday about the release and concentrations of PCB's in the environment in 1975 is that most of what we have learned since 1972 is reasonably consistent with the very crude model that Sarofim and I constructed in 1972. That is not to say that the model was right. All it says is that our ignorance about the exact rates and routes of transport of PCB's in the environment is nearly as profound in 1975 as it was in 1972.

I have found only one area where there does seem to be substantial quantitative discrepancy between our 1972 model and the data generated subsequently; that is, the numbers reported to us here and published in the interim,[†] on concentrations in air and air transport. We have heard some numbers of the order of 200 parts per trillion of PCB's in snowmelt, reports of numbers of the order of 100 nanograms per cubic meter for concentration of PCB's in air, and the number of the order of 70 – 200 and locally up to 2,000 nanograms per square meter per day for rates of dry deposition. These are all somewhat higher than we anticipated on the basis of our model.

If we extrapolate up on the back of an envelope basis from 200 nanograms per square meter per day over the whole continent, that comes out to be something of the order of 1,000 tons. Similarly, if you extrapolate up from 100 parts per trillion in rainfall over the entire continent, you get something of the order of 1,000 tons.

Now, it may be that the numbers we heard were biased toward urban areas, where the concentration of PCB's may be higher than they are in the United States as a whole. Even so, we are now being given numbers that suggest that at least 1,000 tons of PCB's per year are falling out onto the terrestrial environment of the United States in rain and particulate matter. That is about as large as numbers we had envisaged for total releases of PCB's into the air in 1971. What makes the problem worse is that nearly all the PCB's that are found in dry fallout, in the air, and in precipitation look like Aroclor 1254, whereas we had thought that only about half of the materials released into the environment even in 1971 would be Aroclor 1254, and a good deal of them would be Aroclor 1242.

So it seems to me that aerial releases and aerial transport are rather larger than anything we could account for according to our knowledge of the uses and releases that we surveyed in 1972. We need to find out where all this material in the air resembling Aroclor 1254 is actually coming from.

One possibility is that it may be coming from leaks from supposedly sealed uses. Perhaps we ought to be looking at transformers to see how much PCB's vaporize from them.

I would now like to go to the six questions which I posed at the beginning of this session and see what we have learned in the course of this conference toward solving these outstanding problems.

1. What happens to chlorinated dibenzofurans in the environment? I think the answer is that still we know almost nothing. We know they are being released into the environment, since they are present in small quantities in commercial PCB's. Dr. Kuratsune in his presentation yesterday indicated that they are actually formed in the environment during use, and Dr. McKinney's presentation suggested they might be formed by metabolism

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[†] Persson, B., *Ornis Scandinavica*, 2:127-135 (1971); Södergren, A., *PCB-Conference II 1972*:15-18 (National Swedish Environment Protection Board, 1973); Bengtson, A. A. and A. Södergren, *Ambio*, 3:84-86 (1974); Munson, T. O., Ch. 6 in *Upper Bay Survey* (Westinghouse Electric Corporation, Final Report to the Maryland Department of Natural Resources, in press).

under certain circumstances. On the other hand, Dr. Risebrough's presentation suggested that they are not retained in the bodies of birds even when they are ingested; that conflicts with Dr. Kuratsune's information that they are retained extraordinarily efficiently in human tissue.

2. What happens to PCB's in dumps? Again, we know almost nothing. Some numbers were mentioned yesterday for water leaking out of dumps; I did some back-of-the-envelope calculations which suggested that the quantities of PCB's escaping are not yet very significant. One is talking about quantities of the order of hundreds of kilograms per year nationwide—certainly not more than a few tons.

3. Are PCB's in sediments going to be covered up or are they going to be continually recycled? There have been some hints in what has been said today that they are going to be recycled. The information from Ms. McDermott about PCB's in areas where there is shipping activity, and information from Dr. Munson about the movement of PCB's in suspended sediments around Chesapeake Bay suggests that these PCB's in shallow waters are not going to stay there and be covered up and disappear. They are going to be with us for a long time into the future.

4. Is it correct that tetrachloro-biphenyls and lower chlorinated species are rapidly degraded and is there significant human exposure to these lower species? I think that we have had very adequate evidence here that tetrachloro isomers are indeed degraded rather rapidly, but not as rapidly as we would like. Dr. Sanborn's presentation just now suggested that in fish the major difference in the efficiency of uptake, retention, and metabolism comes between trichlorobiphenyls and tetrachlorobiphenyls, and that fish do indeed retain tetrachlorobiphenyls fairly efficiently. That was supported by data from the National Fish Monitoring Program, which suggested that a substantial fraction of the PCB's in the fish look like Aroclor 1242 and 1248; that means that they include substantial quantities of tetrachlorobiphenyls. This in turn implies that humans are exposed to tetrachlorobiphenyls. Although the tetrachlorobiphenyls appear to be rare in human tissue, what that tells us is that humans do not retain tetrachlorobiphenyls. It does not tell us that they are not exposed to them.

This has some important implications from the regulatory point of view. We may have to consider, for example, whether Aroclor 1016 is environmentally "safe," in the sense that modest releases can be regarded as acceptable. These data indicate that releases of Aroclor 1016 into water will lead to human exposure, at least to tetrachlorobiphenyls.

5. What is the extent of human exposure? The data derive from two sides, monitoring of human tissue and monitoring of human food. Human tissue monitoring tells us that human exposure is very widespread. Human food monitoring tells us that most PCB's in human food are in fish.

Unfortunately, that makes it very difficult to estimate human exposure quantitatively, because we know from the fish monitoring program that the levels of PCB's in fish are extraordinarily variable in space. Despite the large number of samples that have been taken by the Canadian program and by the FDA, it is very difficult to define an average exposure. I would go so far as to say that an "average" exposure is a meaningless concept, because some individuals who live in areas where fish residues are high will get exposures 100 times greater than the average, whereas those persons who live in areas where there is little contamination, or who do not like fish, will get very little.

Nevertheless, we do have some numbers for human dietary exposure. The Total Diet Program in the United States gave a figure of the order of 9 micrograms per day for the intake of the average adult. I also did some calculations from Mr. Graham's figures on the Canadian commercial fish to see how many kilograms of PCB's were brought in in Canadian commercial fish and what fraction was eaten. That led me to a figure around 5 micrograms per day for the average intake of the average person in the Canadian population. We have to remember that the average adult does not eat much fish.

Unfortunately, these figures are quite misleading because it is very easy for an individual who is a sports fisherman and who catches salmon, or trout, or chub to take in very much more than that. One calculation in our 1972 paper indicated that it would be very easy for a fisherman or a member of his family to average 300 micrograms per day: this would represent about 4 micrograms per kilogram per day for an adult.

One important point is that exposure of breast-fed infants is likely to be very much greater than that of any adult, even an individual who likes fish. Although Dr. Kutz did not give numerical estimates of PCB levels in human milk in this session, earlier data suggest that a typical level would be around 30 parts per billion.* This leads to an estimate of 4–5 micrograms per kilogram per day intake for the average breast-fed infant. There would

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*PCB levels reported in human milk have been in the range 10–100 ppb: Risebrough, R. W., and V. Brodine, *Environment* 12:16-27 (1969); Westöö, G., Norén, K., and M. Andersson, *Vår Föda*, 22:10-31 (1970); Acker, L., and E. Schulte, *Naturwissenschaften*, 57:497 (1970); Savage, E. P., Tessari, J. D., Malberg, J. W., Wheeler, H. W., and J. R. Bagby, *Pesticides Monitoring Journal*, 7:1-5 (1973). A concentration of about 30 ppb would correspond to the median level of about 1 ppm reported in human fat.

be many above the average and many below.

Accordingly, when considering averages, we should remember that certain individuals are going to get 10 to 100 times the exposure of the average person, as calculated from an average diet.

6. The most complicated question posed in 1972: what would be the effect of Monsanto's curtailment of dispersive uses? A number of speakers yesterday and today have talked about decreases of PCB levels in certain areas or compartments of the environment. For example, there have been decreases in the total dietary intake estimated from the FDA Total Diet Program, in the frequency of findings of PCB's in milk and fish in the FDA's surveillance samples, and in runoff into the Southern California coastal waters. On the other hand, we have also heard about some levels that have not changed, particularly levels in fish in Lake Michigan.

There are three reasons why the curtailment of sales by Monsanto in 1971 should not have been expected to lead to an immediate reduction in environmental levels. One is that not all the closed system uses are actually closed. There are some losses that we heard about today, even from transformer and capacitor uses, which are systems as closed as we are likely to get. Secondly, and more important, there is still a large backlog of products containing PCB's, that were in service prior to 1971, which are still in service and are still being discarded and still leaching. Some of the users of hydraulic fluids, heat transfer fluids, compressors, and so on, are probably still using PCB's that they had several years ago. Certainly the scrapping of capacitors and any losses that may result from disposal of transformers are going to lead to releases for years into the future.

Finally, there is an environmental inertia: it takes a long time after the input into the environment is cut off for the levels in fish, for example, to go down. The time lags will depend on the part of the system under consideration; it is evidently quite short for mussels in Southern California, but is longer for the fish in the same area. Time lags have been quite short for the starlings, which reflect terrestrial uses, and for soil residues. The time lag is likely to be long for somewhere like Lake Michigan, which is a closed system, with a large reservoir of PCB's in the sediment. So I think we should not have hoped for a very rapid decline in PCB's since 1971.

On the other hand, it has been somewhat disappointing that it has been so difficult to find any change in the first 3 or 4 years.

One of the longest time lags of all is likely to be in human tissue, because there is evidence that PCB's are retained for an extremely long time in human tissue. We will probably have to wait for some considerable time after PCB levels have declined in fish before we see a significant decline in human tissue residues.

GENERAL DISCUSSION OF SESSION III

DR. JOSEPH HIGHLAND (Environmental Defense Fund, Washington, D.C.): I want to ask Dr. Jelinek about the survey for fresh fish. Were there many differences in the last several years in the program which might lead to differences in the levels of the species. I was wondering if you could tell us a little bit more about that. I am not very clear on what differences you are talking about.

DR. CHARLES JELINEK (Food and Drug Administration, Washington, D.C.): First of all, one of the big differences is in the fact that all of our activities included compliance followups when we found any species or areas that had high levels. And we are going to dig in on that to find out just how much of a problem it is.

Obviously, from a statistical standpoint, that would throw things way out of whack. You are not getting representative sampling. That is one big difference, in the years where you have a higher proportion of that, you are apt to have a higher proportion of violative samples.

In our comprehensive fish survey program, which we carried out in 1973 and half of 1974, we included saltwater and freshwater fish. So again, you are probably going to have pretty low frequency of high findings, because we did pick up a lot of saltwater fish there. In our regular pesticide surveillance program, that is carried out more within the confines of the United States, although there are some coastal water fish that might be picked up, so that would sort of be in between these two activities. Where you try to combine all these together, it is just impossible to decide, at least with the data that we have, whether there have any changes.

DR. JAMES R. SANBORN (Illinois Natural History Survey, Urbana, Illinois): Our national monitoring program does look at three types of fishes, the predator, and one intervening. Locating the fish in each sampling does create a problem. So we get a cross section of the fish's species. We do notice that many species have a much higher fat content than others. And even if there is a variation within those species as to their body conditions, you would find a correct relationship in the amount of fat and the age of the fish. Consequently, when we look at those fish–lake trout can exist in those environments within a period of time and gather a rather substantial amount of adipose tissue. The goldfish, for example, is a very fat fish. You will find it loaded with PCB's and pesticides.

MR. DONALD TESLACK (EPA): I'd like to find out if there is any data–how does 99 percent average?

DR. JELINEK: I don't believe I have that figure here, and I would hate to speak from memory, sir. If you want to, you know, you can leave me your name and I can see–

DR. IAN C. NISBET (Massachusetts Audubon Society, Lincoln, Massachusetts): Speaking from memory, I believe it would be 95 percent.

DR. ROBERT GOLDEN (Environmental Protection Agency, Washington, D.C.): I understand that fishmeal is used to feed poultry extensively. Is there any information available possibly on secondary exposure to humans?

DR. JELINEK: Well, these levels that we are finding now in poultry, or rather USDA is the one who monitors poultry, are exceedingly low. In the one slide there, the 1 percent of poultry that the findings were on were down around 1 ppm or something like that.

Also, I would say this; our tolerance for PCB's in finished animal feed is two-tenths of a ppm, which is just above the limits of our analytical methods for accurate reproducible quantitative results of animal feed. So we set the tolerance there at almost what you would call the analytical nondetectable method for enforcement purposes.

20 November 1975

Session IV:

ECOLOGICAL EFFECTS AND EXPOSURE

Donald I. Mount, Ph.D.*
Session Chairman

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*Director, Environmental Research Laboratory, Environmental Protection Agency, Duluth, Minnesota.

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INTRODUCTORY REMARKS

Donald I. Mount, Ph.D.

Not being a back-of-the envelope modeler like Dr. Nisbet, I am not going to try to play the role that he did. But I do think there are two or three very quick comments that might help in moving into our discussion.

First of all, it is worth pointing out that there are three primary reasons to be concerned about the impact of pollutants on aquatic organisms. First and most obvious are the effects of pollutants on the organisms themselves. Perhaps less obvious is the warning that when residues accumulate in aquatic organisms, potential hazards to human health may exist.

We have heard about the impact of residues in human food on humans. I think it is worth emphasizing that essentially 100 percent of the organism's diet has residues unlike those in humans. Therefore, the significance of a given concentration of PCB's in the food of an animal such as the mink may be quite different than it is to a human.

I had talked with Dr. Nisbet before the conference and we agreed in the upcoming session to discuss residues only where they relate to effects on the organisms carrying them.

I have noted as I have sat in the audience that it has been very difficult to tell whether the speaker is saying parts per billion or parts per million. So I have asked our speakers if they can refer to micrograms per liter and milligrams per liter. For those who cannot make the conversion, that is parts per billion and parts per million, respectively. So, if a speaker says milligrams per liter, that is parts per million.

I would like now to start with our papers and we would like to reverse the position of the first two papers, because the first one is to deal with the information that was available up to 1972 regarding the effects of PCB's in nonhuman organisms. We are referring, of course, to those organisms in the environment more so than domestic animals. If you wonder why 1972 was picked, it correlates roughly with the report of the PCB task force.

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SUMMARY OF RECENT INFORMATION REGARDING EFFECTS OF PCB's ON BIRDS AND MAMMALS

Rey C. Stendell*

Abstract

The significance of PCB's to wild animals depends both upon their lethal toxicity and their sublethal physiological effects. The lethal dietary toxicities of most Aroclors to experimental birds generally are less than those of DDE, DDT, or dieldrin. Effects of PCB's on reproduction are apt to have the most serious impact on populations. These effects are difficult to evaluate. The chief reproductive effects in chickens are reduced egg production and hatchability. Deformities in chicks are common, and growth rates of young sometimes are depressed. Administration of low dietary levels of PCB's to chickens, bobwhite quail, ringdoves, and mallards have not resulted in thinning of eggshells. No tests of shell thinning have been made with birds of prey or other critical species. Residues of PCB's in wild birds often are at levels known to have caused reproductive impairment in chickens. Species differences must be considered, however, and the significance of these levels to wild birds is very uncertain.

PCB's have been implicated in the reproductive failure and mortality of mink from the Great Lakes region. High levels of PCB's and organochlorine pesticides have been found in marine mammals and have been linked with premature births in some species. Recent studies have shown that some species of bats may be sensitive to a low level of PCB contamination.

INTRODUCTION

The widespread distribution of PCB's in the environment and their effects upon populations of wild birds and mammals are matters of growing concern. Their presence has stimulated research to evaluate their role in the biosphere. This paper reviews recent literature (1972-1975) on the effects of PCB's upon birds and mammals.

EFFECTS ON BIRDS

Lethal Toxicity

Outright mortality can affect populations. Measurement of direct toxicity is an important first step in the evaluation of a chemical. Toxicities of different PCB's to young pheasants (*Phasianus colchicus*), mallards (*Anas platyrhynchos*), bobwhite quail (*Colinus virginianus*), and coturnix quail (*Coturnix coturnix*) have been compared with the toxicities of DDT, dieldrin, and other insecticides (ref. 1). LC_{50}'s for the PCB's were high. Tests of six PCB mixtures, containing 32 to 62 percent chlorine, showed that the toxicity increased with the percentage of chlorine. The dietary toxicities of the Aroclors were, with few exceptions, less than those of the four organochlorine pesticides used for comparison. Special tests with coturnix quail showed that the toxic effects of DDE and Aroclor 1254 were additive but not synergistic. In other studies, Aroclor 1254 was approximately as toxic as DDE to four species of blackbirds (ref. 2). Redwinged blackbirds (*Agelaius phoeniceus*) were somewhat more susceptible to DDE than to PCB's.

Regular oral doses from 10 to 210 mg Aroclor 1254 produced some mortality among subadult pheasants (ref. 3). The amount and timing of the mortality were related to the dose. Heavier birds lived longer and lost the greatest percentage of their body weight before death.

Residues of PCB's in brains of blackbirds given heavy doses of Aroclor 1254 were diagnostic of death (ref. 4); residues in brains of birds that died varied from 349 to 763 ppm (wet weight) and were not above 301 ppm in survivors. In studies with pheasants given daily doses of 210 mg of Aroclor 1254, a brain residue level of 300 to 400 ppm generally indicated death due to PCB toxicosis (ref. 3). Residues of PCB's in livers and other tissues were more variable and of lesser value for diagnosing death.

PCB residues in brains of bald eagles (*Haliaeetus leucocephalus*) found dead from various causes in the United States between 1969 and 1972 varied from 0.10 to 230 ppm (refs. 5,6). Even the highest levels in the eagle brains were below the lethal range determined with captive blackbirds. However, there may be at least two distinct modes of death from PCB's (ref. 7). A sudden heavy intake may cause death from neurotoxicity, with brain residues high and diagnostic of death. Long, low intake, which may occur in the field, may kill by causing edema and related phenomena; signs vary between individuals and between species. Under these circumstances, mortality resulting from PCB's would not necessarily be accompanied by high residues of PCB's in brains, and for these birds no good diagnostic technique is available, for the signs are nonspecific and the residue levels vary greatly.

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PCB's were implicated in a dieoff of guilemots (*Uria aalge*) which occurred in the Irish Sea region in 1969 (ref. 8). The body load of PCB's in birds that died was about twice that in healthy birds collected in the same general region. Moreover, livers of shot birds contained only 0.9 percent of the total body burden compared to 22.5 percent for birds found dead. PCB residues in brains were not determined.

Uptake and Loss Rates

PCB's are readily taken up by animal tissues, and loss rates are reasonably slow. Like DDT and other lipid-soluble pesticides, PCB's accumulate in adipose tissue and have been shown to move out of adipose tissue during lipid mobilization. In experimentally dosed birds, highest PCB levels accumulated in adipose tissue, followed by kidney, liver, brain, muscle, and blood (refs. 3,9).

In experimentally dosed grackles (*Quiscalus quiscula*), half of the PCB's were gone from the tissues in about 1 month, compared to an estimated 6 months for DDE (ref. 4). Continued loss of PCB's after the first month, however, was exceedingly slow and there was no significant further loss occurring during the next 12 weeks. After 32 weeks, about 15 percent of the original burden of PCB's remained in the body.

PCB's are readily excreted in the egg. PCB levels in the eggs of double-crested cormorants (*Phalacrocorax auritus*) reflected carcass levels, but this relationship did not hold for white pelicans (*Pelacanus erythrorhynchos*) (ref. 10). In ringdoves (*Streptopelia risoria*), Aroclor 1254 fed at 10 ppm reached an equilibrium level of 17 ppm wet weight in the eggs; this was about 2 percent of the level found in the fat of the adult birds (ref. 9).

PCB's stored in the adipose tissue are readily mobilized during periods of stress that cause loss of body weight. Alternate starving and feeding of pheasants dosed with PCB's increased the toxicity of the PCB's (ref. 11). Birds that died from starvation had mobilized their fat reserves and had considerably higher PCB's in the brain and other tissues than did PCB-treated birds that were not starved. Similarly, increased levels of PCB in muscle, brain, and liver of PCB-treated ringdoves followed depletion of fat reserves by starvation. At death, PCB concentration in the brains of starved birds had increased 56-fold (ref. 9).

Reproductive Effects

Effects of PCB's on reproduction are difficult to evaluate but are apt to have the most serious impact on populations. Few data are available for wild species, so work done with chickens must be relied upon. Numerous good studies have shown that chickens are sensitive to PCB's. The chief reproductive effects in chickens are lowered egg production and reduced hatchability. Deformities in chicks are common, and growth of young may be depressed. Survival of young sometimes is affected. Feed consumption, adult survival and body weight, eggshell characteristics, and gametogenesis usually are not affected. Most embryo mortality occurs during the latter stages of incubation, although this may depend upon the PCB residue in the egg. Many chicks may die after pipping. The most severe effects are produced by Aroclors in the middle of the range—from 1232 through 1254.

Chickens fed 2 ppm of several Aroclors in the diet for 9 weeks were not adversely affected (ref. 12). Others fed 20 ppm were affected differently by the different types of Aroclors. Aroclors 1221 and 1268 did not affect egg production or hatchability. Aroclor 1248 produced the most severe effects, causing some mortality of adults and nearly eliminating hatching of eggs. Aroclor 1242 produced effects that were very nearly as severe. Aroclor 1232 produced less effect.

The lack of effects of 2 ppm of dietary PCB on chicken reproduction was supported by a 39-week feeding test with Aroclor 1254 (ref. 13). Five ppm of 1254 reduced egg production erratically over the 39-week period. With 50 ppm, however, mortality began, and dosage was stopped after 14 weeks. Egg production fell sharply. Hatchability dropped nearly to zero; residues in eggs were then about 25 to 50 ppm. As residues in eggs dropped after 6 weeks of clean food, hatchability rose. Residues over 10 to 15 ppm in eggs were accompanied by heavy embryotoxicity, but those below 5 ppm showed no effect. At the start of the 50-ppm dosage, deaths of embryos came late in development, but as residues built up deaths came progressively earlier.

The effects on reproduction were further investigated in another study in which chickens were given 50 ppm Aroclor 1254 in water for 6 weeks, and then were given untreated water for 20 weeks (ref. 14). Hatchability dropped to zero within 3 weeks after dosage began and stayed nearly at zero through 8 weeks of clean water, then rose to approximately normal levels after 16 weeks of untreated water. The amount of embryonic mortality associated with a given amount of PCB residue in the egg became higher as the study progressed. Thus, 50 percent mortality of embryos was correlated with 50 ppm of PCB in the yolk after 1.6 weeks of dosage, but with 10 ppm after 18.7 weeks, a fivefold difference. The greatest toxicity per unit of PCB came after 11 weeks of untreated water. Late in the study, 6 to 8 ppm of PCB in yolks was correlated with 14 to 36 percent mortality of embryos. This would represent about 3.6 ppm in the whole egg. The authors concluded that the increase of toxicity with time was caused by the accumulation of some persistent isomer or homolog of 1254 or by a metabolite.

In another study with chickens, egg production was reduced by 10 percent and hatchability by 44 percent when eggs from hens dosed with Aroclor 1248 contained only 3 ppm (ref. 15). When levels in the eggs reached 4.5 ppm, production was further reduced and hatchability was almost completely eliminated.

PCB residues in eggs of wild birds have often equalled or exceeded the levels known to have caused reproductive problems in chickens. For a few examples, 11 eggs of bald eagles from the United States contained from 2.2 to 27.7 ppm of PCB's with a median of 9.7 ppm (ref. 16). PCB residues in osprey (*Pandion haliaetus*) eggs collected in Connecticut in 1968 and 1969 varied from 3.6 to 51 ppm (ref. 17) and brown pelican (*Pelecanus occidentalis*) eggs from the Eastern United States contained from 1.9 to 36.5 ppm PCB's, with many readings over 5 ppm (ref. 18). Species differences in response must be considered in the interpretation of the effects of these levels upon reproduction of wild birds. The number of species that may respond like chickens is not known.

PCB's caused deformities in chicks from hens dosed with 50 ppm Aroclor 1254 (ref. 19). Many deformities appeared when residues in yolks were 10 ppm or more. In another study in which hens were dosed with 20 ppm of Aroclors 1232, 1242, 1248, or 1254, 34 percent of 843 embryos that died during incubation showed signs of abnormal development (ref. 20). PCB's or related chemicals were suspected of causing deformities in a small number of young in a tern colony in which PCB residues were high (ref. 21).

Other species of birds may be less sensitive to PCB's than chickens. Mallards showed no decline in reproductive success during 2 years of dosage with 25 ppm of Aroclor 1254 (ref. 1). Similar results have been reported for coturnix quail (refs. 15,22) and bobwhite quail (ref. 1). Pheasants given oral doses of Aroclor 1254 showed depressed egg production and hatchability, suggesting a response similar to that of chickens (ref. 23).

Decreases in eggshell thickness and associated declines in populations of certain species of wild birds have been linked with elevated levels of DDE in their eggs (for review see ref. 24). The effect of DDE upon eggshell thickness has been demonstrated experimentally with several species (ref. 24). Recent experimental studies with PCB's, however, have failed to demonstrate that low dietary dosages cause significant eggshell thinning in mallards (ref. 1), bobwhite quail (ref. 1), pheasants (ref. 23), ringdoves (ref. 25), or chickens (refs. 12,15,26,27). In earlier tests, chickens fed Aroclor 1242 at 10 ppm or 100 ppm and Aroclor 1254 at 100 ppm laid eggs with thinner shells than did controls (ref. 28). Coturnix quail hens given 10 ppm Aroclor 1242 in the feed for 40 days produced eggs with shells 5 percent thinner than did hens given untreated feed (ref. 29). Similarly, 50 ppm of Aroclor 1254 in the diet produced a small decrease in shell thickness and an increase in the percentage of cracked eggs of coturnix quail (ref. 22). No tests of eggshell thinning have been made with birds of prey or other critical species.

Statistical evaluation of the role that different chemicals may play in thinning the eggshells of brown pelicans in the field has shown that DDE residues correlate much better with shell thinning than do residues of PCB's (ref. 18). Similar relationships have been shown for other species (ref. 30).

Effect on Chromosomes

Few cytogenetic studies have been conducted to determine effects of PCB's on chromosome structure. In one study, no chromosomal aberrations were detected in chicken embryos following injection of Aroclor 1242 into the egg to levels of 10 and 20 ppm (ref. 31). At these levels mortality of embryos was high. In another study, embryos from ringdoves treated with Aroclor 1254 at 10 ppm had a higher frequency of chromosome aberrations than did controls (ref. 32).

Chromosomal aberrations may contribute to the abnormalities of developing embryos observed in some studies, or perhaps to a decreased reproductive success during successive generations of exposure to PCB's. When ringdoves were exposed to 10 ppm Aroclor 1254 during two generations, the reproductive success of the first generation was normal, but the success of the second generation was greatly reduced (ref. 32).

Effects on Behavior

Alterations in behavior may bring about depressed survival and reproductive success of wild species. Experimental studies have shown that PCB's may alter behavior. Caged European robins (*Erithacus rubecula*) fed PCB's showed increased migratory activity compared with untreated controls (ref. 33). PCB's caused a similar tendency in redstarts (*Phoenicurus phoenicurus*) (ref.

34). Behavior, on a visual cliff, of pheasant chicks hatched from hens given 50 mg Aroclor 1254 weekly was significantly different from controls or those receiving 12.5 mg (ref. 23); more chicks from the 50-mg group jumped to the visually deep side or made no choice of sides than chicks from other groups. The avoidance response to a moving silhouette of coturnix quail chicks fed 200 ppm of Aroclor 1254 was greatly reduced (ref. 35); there was no significant recovery in response after the birds were again given clean feed, suggesting an effect upon the maturing central nervous system.

Embryonic mortality of ringdoves fed 10 ppm Aroclor 1254 was higher when the eggs were incubated by the parent birds than when they were incubated artificially (ref. 36). Egg temperatures suggested that the increased mortality was due to decreased parental attentiveness.

EFFECTS ON MAMMALS

Certain species of mammals appear to be especially sensitive to ingestion of low levels of PCB's. An extreme example was provided by the declining reproductive success and increasing mortality of mink that was observed by commercial mink ranchers in the Great Lakes region in the 1960's. The problems developed when mink ranchers began to use coho salmon from the Great Lakes in mink rations (ref. 37). Other fish from Lake Michigan had similar effects, but coho from the Pacific coast caused no trouble. Feeding trials were conducted with several contaminants that had been identified in the fish (ref. 38). Neither DDT nor dieldrin caused these effects in mink, even at levels far higher than those in the fish. In the first test, a mixture of PCB's at 30 ppm in the diet killed all the adult mink. With dietary dosages of 5 and 10 ppm of Aroclor 1254, no reproduction occurred. At 10 ppm, five of the six adults died. In further tests, 1 ppm of PCB in the diet reduced reproductive success. More drastic results were observed when the mink were fed meat from cows that had been dosed with Aroclor 1254 (ref. 39). When the concentration of PCB in the diet was 3.57 ppm, there was no reproduction and all breeders died. When the concentration was 0.64 ppm, some adults died and no surviving young were produced.

High levels of PCB's and organochlorine insecticides have been found in marine mammals and a link with premature births in some species has been suggested. Premature pups from California sea lions (*Zalophus californicus*) contained higher levels of these compounds in the blubber, liver, and brain than did full-term pups (ref. 40).

Recent studies have shown that certain species of bats are sensitive to PCB's. In big-brown bats (*Eptesicus fuscus*), PCB's crossed the placenta 2 to 3 times more readily than DDE (ref. 41). Concentrations of PCB's were significantly greater in litters that included dead young than in litters in which both young were born alive. In further studies, big-brown bats fed 250 ppm DDE in the diet gained weight and none died, but individuals fed only 10 ppm Aroclor 1254 gained weight more slowly and some died (ref. 42).

PCB's have been shown to be toxic to some nonhuman primates over a wide dose range. Adult rhesus monkeys developed signs of PCB intoxication within 1 to 2 months at doses as low as 2.5 and 5.0 ppm of Aroclor 1248 in the diet (ref. 43); these levels also caused a marked decline in reproductive success.

CONCLUSIONS

The effects of PCB's upon wild populations are difficult to evaluate and many important questions remain to be answered. Residues in wild species may be derived from different commercial mixtures that have undergone metabolic changes for varying time periods before reaching the target organism. Residues of PCB's in wild birds often are at levels known to have caused reproductive impairment in chickens. Nevertheless, species differences must be considered and it is not known how many species respond in the same manner as chickens. The great sensitivity of mink raises the question of how many other mammals may be equally sensitive to reproductive impairment or mortality.

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PRE-1972 KNOWLEDGE OF NONHUMAN EFFECTS OF POLYCHLORINATED BIPHENYLS

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Abstract

Research conducted worldwide on the effects of PCB on aquatic organisms, birds, and mammals is herein summarized. The ecological effects of PCB are still not fully understood, nor is the long-term impact of PCB on animal populations. It is known that PCB are highly persistent contaminants that bioaccumulate in the food chain. They produce both chronic and acute effects on the growth, reproduction, and behavior of fish, birds, and mammals. Of these, freshwater fish accumulate PCB to the greatest extent. The birds and mammals that feed on freshwater fish in turn take in hazardous amounts of PCB residues and become subject to their serious toxicological effects.

INTRODUCTION

Polychlorinated biphenyls were under investigative development as early as 1881 (Schmidt and Schultz) and yet very little knowledge of their toxic effects was evident until they had gained wide use during the 1930's and pathological studies on laboratory animals were conducted (refs. 5,41). These polychlorinated biphenyls, napthlenes, terpenes, etc., were marketed under a variety of trade names–Aroclor, Chlorphen, Kanechlor, Phenochlor–and a series of numerical synonyms such as we have become familiar with in Aroclors–1221, 1232, 1242, 1248, 1254, 1260, 1268, etc. (refs. 8, 33, 51).

Almost 20 years lapsed until McLaughlin et al. reported that Aroclor 1242 was highly toxic and produced teratogenic effects in chick embryo at low dosage levels (ref. 44). Many studies were initiated at about this time, when analytical chemists monitoring environmental components observed a number of unidentified peaks in gas chromatographic analysis that were most frequently associated with aquatic species of birds and fishes (refs. 3, 19, 20, 21, 22, 24, 25, 26, 27, 32, 33, 34, 45, 46, 48, 53, 54, 55, 56, 58, 59, 61, 64, 66, 67, 68, 83, 84, 85, 86, 88, 93, 94). The annual research reports by the U.S. Fish and Wildlife Service since 1969 gave technical details in the developments of our work on the toxicity of PCB and chlorinated hydrocarbon pesticides, which coincided with monitoring investigations of persistent chemicals such as DDT (refs. 3, 12, 16, 17, 38, 39, 40, 47, 52, 57, 71, 77, 78, 79, 80, 81, 82, 89, 90, 91, 92). The development of the silicic acid method allowed for the separation of PCB from other pesticide components and permitted detailed investigation of these environmental contaminants (refs. 1,71).

The December 1971 conference on polychlorinated biphenyls sponsored by the National Institute of Environmental Sciences at the Quail Roost Conference in Rougemont, North Carolina, provided full array of the effect of PCB on experimental animals, fish, and wildlife. The Fish and Wildlife Service National Pesticide Monitoring Program discussed at this conference concentrations of PCB in fish samples (ref. 89). These residue levels were confirmed in the cross-check analysis by the Fish Pesticide Research Laboratory, which in turn has spurred more toxicological research into the significance of these residues (refs. 18, 40, 71, 74, 75, 76, 80).

EFFECTS ON AQUATIC ORGANISMS

Chronic and acute toxicity studies on aquatic organisms were conducted in which 96-hour LC_{50} values were determined for eight of the Aroclor series from 1221 through 1268 (refs. 40,71). The 96-hour LC values ranged from 1,170 to 50,000 mg/l for cutthroat trout (table 1) (refs. 40,71). In another test, the acute oral toxicities of Aroclors 1242, 1248, 1254, and 1260 were found to be greater than 1500 mg/kg in rainbow trout. Intermittent flow for Aroclors 1242, 1248, and 1254 indicated that these compounds were much more toxic at high temperatures and at longer exposure periods. Comparable results were obtained on channel catfish and rainbow trout (table 1) (refs. 40,71).

The effect of Aroclors on invertebrate forms gave an even wider distribution of effects (table 2) (refs. 40,69,71), with Aroclor 1248 being the most toxic to *Daphnia magna* and levels above 5 mg/l causing inhibition in reproduction (ref. 46). Similar results were obtained on *Gammarus pseudolimnaeus* (ref. 45). The 96-hour LC_{50} values for Aroclors 1248 and 1254 were 52 and 2400 mg/l respectively (ref. 40). Another species of scud (*Gammarus fasciatus*) was found to concentrate PCB by 27,000 times the exposure level compared to the *Daphnia*, which concentrated Aroclor 1254 by 48,000 times (figure 1) (ref. 40).

Both crayfish and the scud were found to be much more sensitive to Aroclor 1242, with the uptake being

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NOTE: Scientific names for fish and wildlife listed in text are given at the end of the paper.

Table 1. Toxicity of PCB to fish (refs. 40,71)

PCB	Invertebrate (species)	Bioassay method[1/]	Exposure time	LC_{50} (µg/g)
Aroclor 1242	Scud (*Gammarus faciatus*)	C	4	10.0
	Scud (*Gammarus faciatus*)	C	10	5.0
	Crayfish (*Orconectes nais*)	A	7	30.0
	Damselfly (*Ischnura verticalis*)	A	4	400.0
	Dragonfly (*Macromia sp.*)	A	7	800.0
Aroclor 1248	Scud (*Gammarus faciatus*)	A	4	52.0
Aroclor 1254	Scud (*Gammarus faciatus*)	A	4	2400.0
	Glass shrimp (*Palaemonetes kadiakensis*)	C	7	3.0
	Crayfish (*Orconectes nais*)	C	7	80.0
	Damselfly (*Ischnura verticalis*)	C	4	200.0
	Dragonfly (*Macromia sp.*)	A	7	100.0
2,4'-Dichloro-biphenyl	Scud (*Gammarus psuedolimnaeus*)	A	4	120.0
4,4'-Dichloro-biphenyl	Scud (*Gammarus psuedolimnaeus*)	A	4	100.0
2,3,4'-Trichloro-biphenyl	Scud (*Gammarus psuedolimnaeus*)	A	4	70.0
2,4,5,2',5'-Penta-chlorobiphenyl	Scud (*Gammarus psuedolimnaeus*)	A	4	210.0
2,4,6,2',4',6'-Hexa-chlorobiphenyl	Scud (*Gammarus psuedolimnaeus*)	A	4	150.0

[1/] A = Acute toxicity by static test procedure
C = Chronic toxicity by flow-through procedure

Table 2. Toxicity of PCB to aquatic invertebrates (refs. 40,69,71)

PCB (Aroclor)	Fish	Temp (°C)	Bioassay method 1/	LC$_{50}$ value in μg/l at exposure time of						
				4d	5d	10d	15d	20d	25d	30d
1221	Cutthroat trout	8.9	A	1170	--	--	--	--	--	--
1232	Cutthroat trout	8.9	A	2500	--	--	--	--	--	--
1242	Cutthroat trout	8.9	A	4530	--	--	--	--	--	--
	Rainbow trout	17.0	C	--	67	48	18	10	12	--
	Bluegill	20.0	C	--	154	72	54	--	--	--
	Bluegill	17.0	C	--	--	--	164	125	120	84
	Channel catfish	20.0	C	--	--	174	107	--	--	--
	Channel catfish	17.0	C	--	--	--	219	150	132	87
1248	Cutthroat trout	8.9	A	5750	--	--	--	--	--	--
	Rainbow trout	17.0	C	--	54	38	16	6.4	3.4	--
	Bluegill	18.3	A	278	--	--	--	--	--	--
	Bluegill	20.0	C	--	307	160	76	10	--	--
	Bluegill	17	C	--	136	115	111	106	100	78
	Channel catfish	18.3	A	6000	--	--	--	--	--	--
	Channel catfish	27.0	C	--	--	94	57	--	--	--
	Channel catfish	20.0	C	--	--	225	127	--	--	--
	Channel catfish	17.0	C	--	--	121	121	115	104	75
1254	Cutthroat trout	8.9	A	42500	--	--	--	--	--	--
	Rainbow trout	20.0	C	--	156	8	--	--	--	--
	Rainbow trout	17.0	C	--	--	160	64	39	27	--
	Bluegill	18.3	A	2740	--	--	--	--	--	--
	Bluegill	20.0	C	--	--	443	204	135	54	--
	Bluegill	17.0	C	--	--	--	303	260	239	177
	Channel catfish	18.3	A	12000	--	--	--	--	--	--
	Channel catfish	20.0	C	--	--	--	741	300	113	--
	Channel catfish	17.0	C	--	--	303	286	293	181	139
1260	Cutthroat trout	8.9	A	60900	--	--	--	--	--	--
	Rainbow trout	20.0	C	--	156	5	--	--	--	--
	Rainbow trout	17.0	C	--	--	326	143	78	49	51
	Bluegill	20.0	C	--	--	--	--	245	212	151
	Bluegill	17.0	C	--	--	--	--	--	--	400
	Channel catfish	20.0	C	--	--	--	--	296	166	137
	Channel catfish	17.0	C	--	--	535	482	512	465	433
1262	Cutthroat trout	8.9	A	50000	--	--	--	--	--	--
1268	Cutthroat trout	8.9	A	50000	--	--	--	--	--	--

1/ A= Acute toxicity by static test procedure.
C= Chronic toxicity by flow-through procedure.

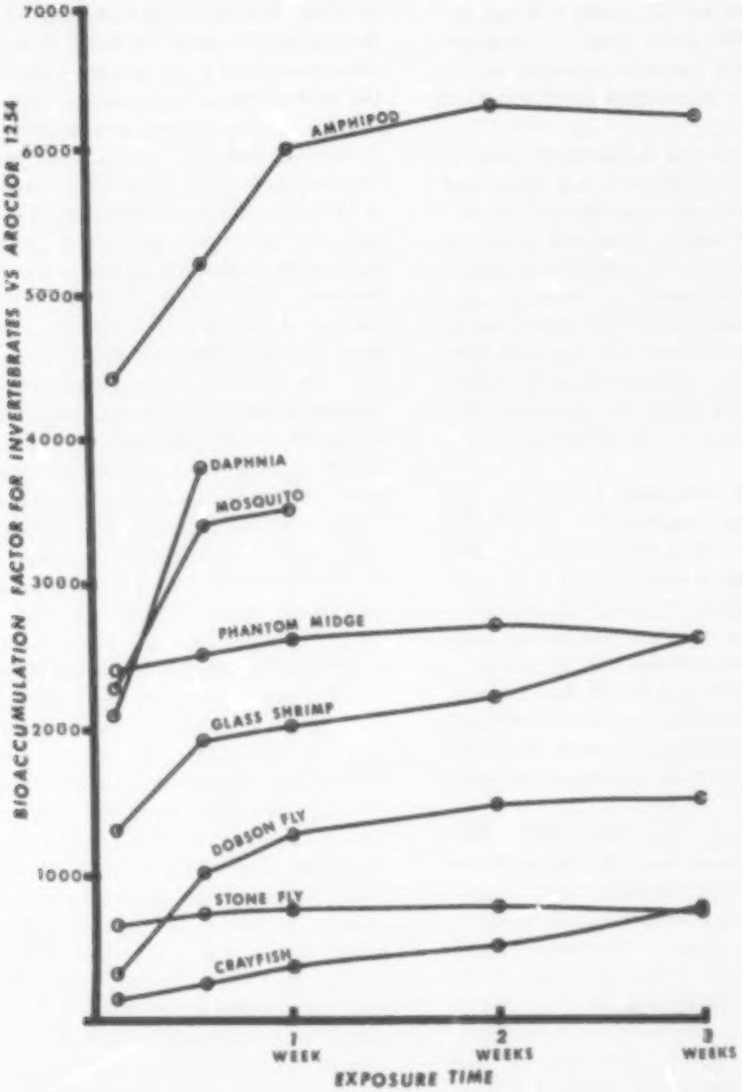

Figure 1. Bioaccumulation of Aroclor 1254 for various aquatic invertebrates exposed to water concentrations: 1.1 μg/l for *Daphnia magna*; 1.6 μg/l for *Gammarus pseudolimnaeus*; 1.2 μg/l for *Orconectes nais*; 1.3 μg/l for *Palaemontes kadiakensis*; 2.8 μg/l for *Pteronarcys dorsata*; 1.1 μg/l for *Corydalus cornutus*; 1.5 μg/l for *Culex tarsalis*; 1.3 μg/l for *Chaoborus punctipennis* (ref. 40).

somewhat enhanced with time. Glass shrimp *(Palaemonetes kadiakensis)* were also found to be quite sensitive to Aroclor 1254, with an LC_{50} value of 3 mg/l in a 7-day exposure. On the other hand, dragon fly (*Macromia* species) and damsel fly (*Ischnura verticalis*) were a great deal more resistant to Aroclors 1254 and 1242 (table 1 and figure 1).

Aroclor 1254 was found to be extremely toxic to immature pink shrimp, of which more than 50 percent died within 15 days of continuous exposure to 0.94 mg/l (ref. 47). A similar concentration factor was noted for Aroclor 1254 in two estuarine fish species (*Lagondon rhomboides* and *Leiostomus xanthurus*). While between 14 and 45 days of exposure to 5 mg/l did product some mortality, the 1 mg/l during the same exposure time resulted in no mortality, but concentration of residues did occur at rates similar to those for freshwater fish—10,000 to 50,000 times the environmental levels (refs. 12,16).

In studies conducted in Sweden on salmon eggs, PCB residues on a lipid basis ranging from 7.7 to 34 µg/g caused mortalities between 16 to 100 percent (ref. 26). The regression analysis gave a coefficient of correlation of 0.85, with a significance of $P = .001$. PCB residues in these Atlantic salmon eggs on a wet weight basis were from 0.4 to 1.9 µg/g. This indicates that the threshold for egg mortality was about 0.5 µg/g PCB. Such residues would be comparable to whole fish residues of 2.5 to 5.0 µg/g or very close to those values found in many fish sampled in our National Pesticide Monitoring Program and in Sweden (refs. 6,89).

The oral toxicities of Aroclors 1242, 1248, 1254, and 1260 were found to be in excess of 1500 µg/kg for adult rainbow trout (ref. 40). However, trout fry from eggs containing 2.7 µg/g of Aroclor 1242 along with a DDT residue of 0.09 µg/g caused a 75 percent cumulative mortality 30 days after hatching (ref. 18). In five other groups of less contaminated eggs, 10 to 28 percent mortality was noted 30 days after hatching. About 60 to 70 percent of the fry that survived were deformed and teratology was evident. Generally speaking, the more chlorinated PCB were not as acutely toxic as the less chlorinated ones. For example, in cutthroat trout, Aroclors 1221, 1232, 1242, 1248, 1254, and 1260 had LC_{50} values for 96-hour exposure of 1.2, 2.5, 5.4, 5.7, 42, and 61 mg/l respectively.

In tests with individual homologs, toxicity decreases as the percentage of chlorine increases in amphipods against 5 PCB formulations (ref. 40). The dichlorobiphenyls and trichlorobiphenyls dominate the Aroclor 1242, whereas the trichlorobiphenyls and tetrachlorobiphenyls dominate in Aroclor 1248 and the pentachlorobiphenyls and hexachlorobiphenyls dominate in the Aroclor 1254. The most toxic PCB formulations appear to be those that contain 4 to 5 chlorine atoms; these predominated in Aroclor 1248, which was found to be the most toxic of all of the Aroclors tested on fish. A noticable shift in residue components following exposure to Aroclor 1254 was observed in experiments conducted on amphipods (ref. 71). An apparent twofold increase in concentration of less chlorinated isomers and homologs occurred in the trichlorobiphenyls and tetrachlorobiphenyls. The higher chlorinated pentachlorobiphenyls and hexachlorobiphenyls were decreased by half in experiments conducted on fish (ref. 40). Tests showed that residue accumulation of PCB in invertebrates ranged from 160 to 6,300 times that in water. In channel catfish, after 77 days of exposure, the concentration of Aroclors 1248 and 1254 accumulated in channel catfish to 56,270 and 61,190 times, respectively, than in water (figure 1). The more chlorinated components accumulated more than the less chlorinated ones, and upon termination of the exposure, PCB elimination by fish was more rapid for the less chlorinated components (refs. 40,71). The researchers also found evidence that the actual uptake is less or the turnover rate is more rapid in the invertebrates. This is in agreement with observations by other investigators (refs. 48, 64, 65, 66, 67).

In other experiments conducted by our Fish Pesticide Research Laboratory, we found that inclusion of Aroclor 1254 in fish food at the rate of 0.448 µg/g resulted in a 52 percent increase in fish thyroid activity. In a high-treatment group that received 480 µg/g, thyroid stimulation was 119 percent over that of the control group (figure 2) (ref. 40). In these tests we found that the lowest detectable stimulation thyroid activity occurred in salmon dosed at 1/1,000th the dosage that would cause mortality. This test was further duplicated in channel catfish with comparable results, which suggests that the laboratory studies may be realistic indicators of the physiological effects of PCB on fish and the aquatic environment (figure 3) (refs. 40,71). The true effects of thyroid stimulation, however, on respiration, carbohydrate metabolism, oxygen consumption, ammonia metabolism, osmoregulation, growth, oxidative phosphorylation, central nervous system function, and behavior of fish are yet to be determined. Nevertheless, we can postulate that these must be important and interrelated factors in the physiological and biochemical function of fish and any alteration of this normal function could effect fishes' ability to adapt to stress or such changes in the environment as salinity and temperature. The effect of environmental stress on the health of fish is very well illustrated by Snieszko (figure 4) (ref. 70). Subtle effects

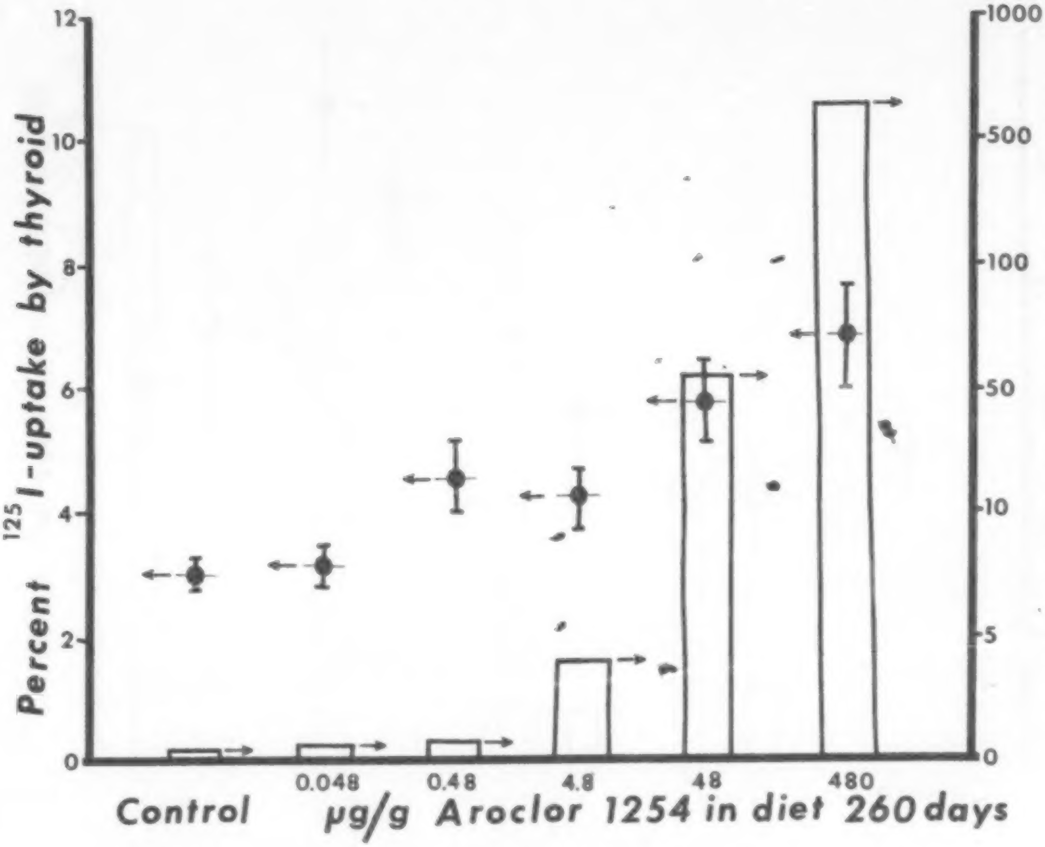

Figure 2. Whole-body residues and thyroidal uptake of ^{125}I in coho salmon fed diets containing Aroclor 1254 for 260 days (ref. 40).

on microflora, fauna, and plankton must also be investigated for potential impacts on trophic level ecology (refs. 29,30).

Enzymatic and compositional changes of the microsomes occur where possible lipids are concentrated in the membrane and manifestation of enzyme malfunction is suspect in the proximity of hydrophobic chlorinated aromatic hydrocarbons such as PCB, DDT, and other organochlorine pesticides that would accumulate (ref. 50). Meyer, Mehrle, and Sanders conducted experiments with incorporation of Aroclor 1248 into the diet of juvenile lake trout in concentrations of 0.2 to 6 mg/kg of food (ref. 40). These levels, comparable to those found in natural food, suppressed growth and serium cortisol levels, but stimulated thyroid activity during the first 160 days of the experiment (ref. 40). After 320 days, however, control and treated fish were similar.

Histopathological examination of fish exposed to Aroclor and chlordane showed a very high incidence of gill lesions after 6 months exposure; 6 percent of the PCB-treated fish were affected, compared to 15 percent after 9 months (refs. 76-78). Progressive and diffuse degeneration changes in liver were noted in 80 percent of the fish after 4 to 6 months exposure to the chlordane or PCB diet. A nonspecific degeneration of liver pyrenchema and cytoplastic vaculation of liver cells occurred in addition to pleomorphism. The severity of liver tissue degeneration throughout the 6- to 9-month exposure period exceeded the controls by at least threefold. After this, we noted that focal areas in liver degeneration decreased in size and by the following year no liver lesions could be observed in this lot of fish. This observation was complemented by the decrease in severity of gill lesions in PCB-treated fish. The gill lesions also improved after 9 to 12 months of exposure and no significant difference between treated and control fish could be detected the following year.

In parallel experiments, whole-body residues had

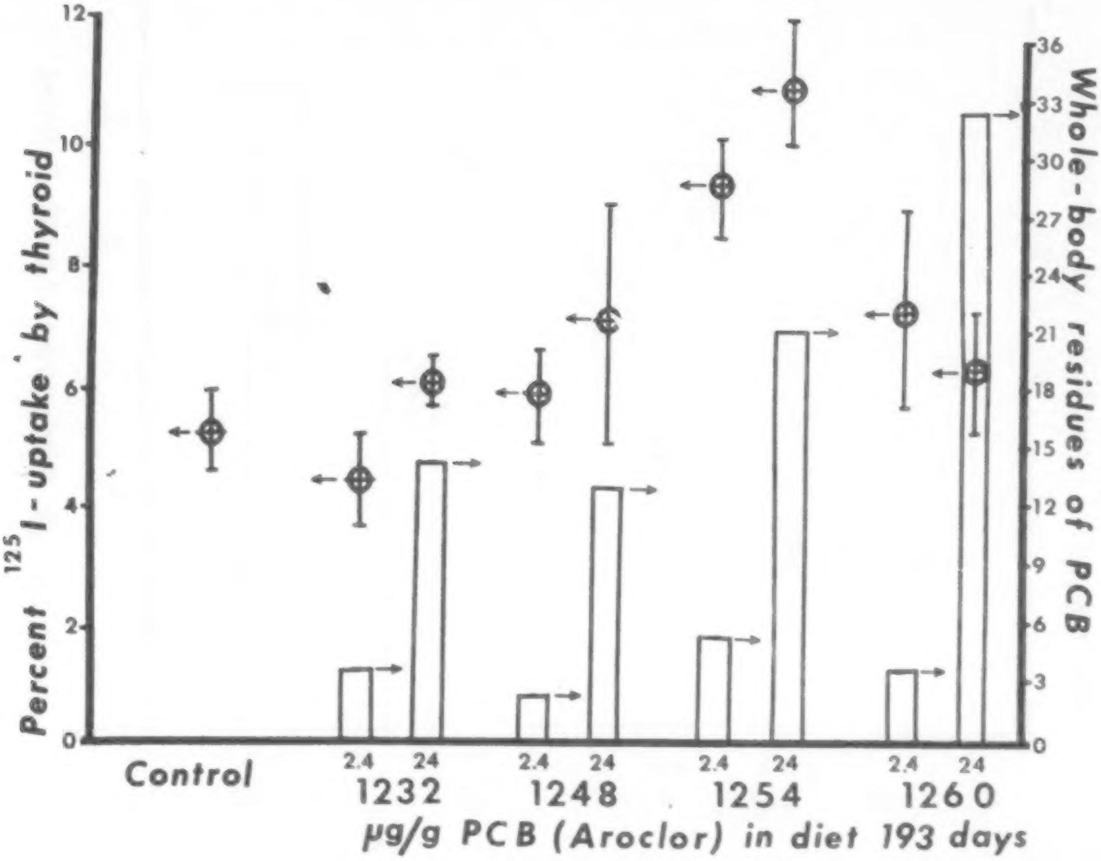

Figure 3. Whole-body residues and thyroidal uptake of ^{125}I in channel catfish fed diets containing Aroclors 1232, 1248, 1253, and 1260 for 193 days (ref. 40).

increased from 0.13 mg/kg at the lower dosage of 0.1 mg/kg in the food to a residue of 9.7 mg/kg at the higher food dosage 6 mg/kg after 320 days (ref. 40). After the trout were fed uncontaminated food for 60 days, the residues declined an average of 31 percent, 15 percent, and 12 percent in the 0.6-, 1.8-, and 6.0-mg/kg treatments respectively. Lake trout growth was retarded by the dietary intake of Aroclor 1248. These results suggest that although fish have a remarkable ability to recuperate and regenerate tissue, the extent and lethality of damage through intoxication by PCB are not clear. However, precise toxicological implication of Aroclors on the general well being and health of fish is certainly suspect (ref. 70).

EFFECTS ON BIRDS AND MAMMALS

Behavioral effects of organochlorine pesticides and PCB have been noted in the studies conducted on Japanese or coturnix quail fed sublethal concentrations and subjected to a simultaneous test for avoidance behavior by rapidly moving away from strange objects (refs. 79,82). This behavior appears in the very early stage of development of galinaceous chicks and is almost certainly essential to their survival in the wild. Seven-day-old coturnix chicks were given Aroclor 1254 in their diet for 8 days and in untreated food for 6 more days. Avoidance behavior was measured daily, and a group avoidance response was significantly suppressed by chlordane,

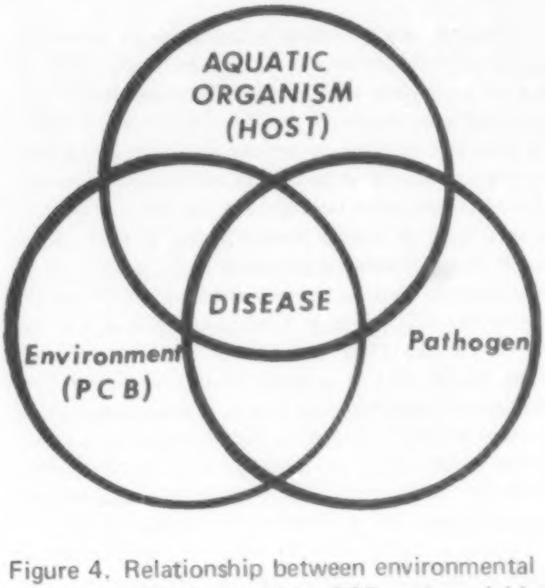

Figure 4. Relationship between environmental stress imposed by PCB and pesticides, susceptible host, and the disease organism as suggested by Sniezko (ref. 70).

dieldrin, and Aroclor 1254, although DDE had no apparent effect. Whereas the behavior of untreated birds returned to normal after 2 days on untreated feed, the response of chicks that were on dieldrin- and chlordane-treated feed improved somewhat during the 6 days of untreated food. However, the response of birds treated with Aroclor 1254 showed no improvement during the period.

Investigative monitoring for pesticide and PCB residues indicated that the highest residues occur in or near urban and industrialized areas. If these data are transferable to many other species of birds, we would expect to see the avoidance reaction suppressed, mortalities would increase from accidents, and predation would be maximized through the encroachment of man on the habitat of the species. The causes of mortality were subjected to autopsy study plus chemical analysis in studies conducted from 1969 through 1970 on 39 bald eagles found dead in various areas in the United States (refs. 42, 57, 79-82). The median residues of DDE in carcuses were 7 ppm in 1969 and 19 ppm in 1970, and mean residues of dieldrin were 0.4 ppm in 1969 and 0.7 ppm in 1970. Six of the eagles had dieldrin residues in the brain of 4.6 to 11 ppm, which is within the lethal range. One eagle contained 385 ppm of DDE and 235 ppm of PCB. In summary, environmental poisons were credited with 18 percent of the deaths, illegal shooting with 46 percent, accidents with 15 percent, natural disease with 8 percent, and miscellaneous and unknown causes with the remaining 13 percent.

In a survey to determine the degree of eggshell thinning and the extent of chemical contamination in aquatic birds in the Texas Coast area, eggs from 21 species were collected in 1971 (refs. 52, 78-80). Chemical analysis completed for 14 species showed that DDT in metabolites existed in all samples, whereas dieldrin residues were found in the Texas Rice Belt and the highest PCB residues were near urban and industrial areas again (ref. 79). The most noticably high PCB residues were found in some eggs of inland feeding species, including: double-crested cormorant (*Phalacrocorax auritus*), which contained 32 ppm; Caspian terns (*Hydroprogne caspia*), 16.5 ppm; and Foster terns (*Sterna forsteri*), 12.5 ppm (ref. 80). However, PCB were present in only 66 percent of the 158 eggs that were analyzed in another study conducted on ospreys (*Pandion haliaetus*) in the Gulf of California. During 1971, the residues of DDT were found to be generally low compared to those in osprey populations showing poor reproductive success in other parts of the range. PCB residues were detected in four of eight eggs and when present were higher than DDE residues; however, eggshell thinning was minor (averaging -3 percent), as expected from their low residues.

An experimental study on the absorption and metabolism of the PCB mixture Aroclor 1254 showed that all the components in the mixture were absorbed in essentially equal proportions from encapsulated dosages (ref. 81). The dietary dosage for quail of 300 ppm of Aroclor 1254 for 14 days followed by 14 or 42 days of untreated food showed that the various components of the Aroclor were excreted at greatly different rates. At the end of the 42 days of clean food, the PCB components changed entirely from the proportions of compounds that were originally fed to birds. Changes in patterns of gas chromatographic peaks that were detected militate against the dependable measurement of the quantities of PCB by gas chromatographic methods that can be related to the environmental or diet intake of the birds.

In another study, the combined effects of p,p'–DDE, and PCB were measured in mallard ducks. The ducks were fed diets containing 40 ppm of each chemical in the diet for 2 months prior to breeding and throughout the reproductive season (ref. 81). Average decreases in eggshell thickness were almost identical between the two compounds, with -17.5 percent for DDT only and -17.0 percent for DDE plus PCB in the diet (refs. 38, 79-80). No eggshell thinning occurred in birds fed PCB alone or in untreated diets except for a slight decrease in shell thickness near the end of the egg laying period. However, the birds fed the DDE-plus-PCB diet layed fewer eggs and egg production dropped off significantly 8 weeks before that of birds that were receiving other treatments. Egg breakage and egg eating were also highest in the DDE-plus-PCB groups and the combination of the chemicals seemed to increase the amount of egg loss through some effect—possibly on behavior. Therefore, overall production problems were most severe when PCB were fed in addition to the DDE.

In the Upper Great Lake States, 9 of 13 species of fish-eating birds were investigated in 1969-1970, and were found to sustain statistically significant decreases in eggshell thickness since 1946 (ref. 82). Maximum changes in thickness index occurred in great blue herons (-25 percent), red breasted mergansers (-23 percent), common mergansers (-15 percent) and double-crested comorants (-15 percent). Great blue heron eggs taken from Louisiana generally displayed smaller changes since 1946 than herons in the Midwest. On a lipid basis, mean PCB and DDE residue levels exceeded 100 ppm in seven of these species in the Lake States and one of seven species in Louisiana. The average DDE:PCB ratios in the two regions were 1.25:1 and 3.9:1 respectively. These ratios contrast with some around 0.3:1 previously reported in oceanic birds in the Bay of Fundy and with ratios of roughly 5:1 and 10:1 that were reported by others on the Pacific Coast. The relationship between shell thinning and DDE content of eggs was apparent for most species, but for herons DDE was the only compound correlated (ref. 52).

Partial correlation carried out on a somewhat questionable family basis showed that PCB also correlated with shell thinning in mergansers, in a study of chlorobiphenyl poisoning with formation of myelin figures in both mouse and monkey liver (ref. 49). Mice were subjected to daily dosages of .001 ml for 12-26 weeks and cornification of smooth surface membranes of the cytoplasmic reticulum was observed (ref. 50). This was also accompanied by a displacement of rough surface membranes and endoplasmic proliferation and an increase in microbodies, lyosomes, and lipid content. Similar observations were made in the squirrel monkey from dosages of 320 mg given over 46 days. Liver enlargement was noted but the main cause of death was attributed to penumonia or diarrhea (ref. 49). A similar experiment was run on the cynomolgus monkey, which received 641 mg in 40 days to 348 mg in 239 days with very similar results (ref 49). Sublethal effects of hepatic enzyme induction were correlated with the breakdown of estradiol and the increase in cytoplasmic RNA in kestrels fed 0.5 to 5.0 ppm of Aroclors 1254 or 1262 for 5 months (ref. 53).

The effects of polychlorinated biphenyls and organochlorine pesticides appear to be manifested in the alteration of enzyme and hormone biochemistry. In unpublished work by Street and his coworkers, Vos reported that in inductions of hepatic microsomal enzymes, the sleeping time of experimental animals was decreased after treatment with hexobarbitol. An enhancement of in vitro rates of aniline hydroxylation and para-nitroanisole demethylations occurred in direct proportion to an increase to chlorine content of different PCB isomers in Aroclors 1221 through 1268 (ref. 86). PCB preparation of Aroclor 1221 and Aroclor 1254 at 1 and 10 mg/kg for 28 days in pregnant rabbits resulted in liver enlargement with increased activity of drug metabolizing enzymes aniline hydroxylase and aminopyrine n-demethylase at the rate of 10 mg/kg of Aroclor 1254, while no effects could be measured at the other levels and with the other levels and with the other Aroclors (ref. 86).

Vos conducted an extensive review of toxicity of PCB for mammals and birds (ref. 86). He concluded that PCB's have several sublethal effects, such as microsomal enzyme induction, porphyrogenic action, estrogenic activity, and immunosuppression. Aroclor 1254 administered to rabbits during the first 28 days of gestation had embryo-toxic effects at levels of 12.5, 25, and 50 mg/kg of body weight, which demonstrates the direct effects on microsomal enzyme activity in pregnant rabbits (ref. 77). In previously cited work by McLaughlin (ref. 44), edema and beak deformities in chicken embryos have been described after yoke sac-injection of 10 and 25 mg of Aroclor 1242 resulted in a 95 and 100 percent embryonic mortality respectively. PCB have also been found to cause neural pathology in rats when administered at a rate of 0.3 to 0.5 ml/kg per day for 14 or 21 days. The pathology is characterized by impairment of motor function and decreased motor velocity with loss of large nerve fibers (ref. 76).

A high dose of Aroclor was required before liver necrosis developed in chickens (ref. 88). Moderate dosages in rabbits produce mottled liver with subacute yellow atrophy and fatty degeneration with marked necrosis at dosage rates of 0.3 and 0.6 g/day, but 0.9 g/day caused death before liver pathology developed (refs. 86,87). Miller (1944) observed 100 percent mortality between 17 and 98 days with similar skin applications of 34.5 mg during an 11-day period caused 100 percent mortality within 28 days while on treatment (ref. 41). Central atrophy of liver cells occurred, with perinuclear basophilic granulation and focal necrosis occurring in a few of the animals.

Nichizumi (1970) utilized light and electron microscopy to confirm the formation of so-called myelin figures in mouse liver associated with a minimum effective dose of 5 mg/kg of a 48% chlorine PCB (refs. 73,88). Japanese quail demonstrated porphyrogenic action when dosed with PCB for 7 days at dosage rates of 1 to 100 mg/kg of Aroclor 1260 (ref. 86). Nichizumi measured the formation of aminoevulinic acid by liver mitochondria; significant increases of greater than 10.5 to 120 m/moles ALA per gram of liver per hour were found. PCB concentrations in the liver ranged from 1.4 to 478 ppm between the 1 mg/kg and 100 mg/kg dosage rates. The most marked effect was noted at the 100 mg/kg dosage rate, in which microscopic tissue fluorescence was seen in three out of five cases and two out of five cases respectively.

Domestic chickens demonstrate an acute susceptibility to Aroclor intoxification; liver enlargement occurs at dosages ranging from 100-1,000 ppm of Aroclor 1242 and mortality occurs at rates above 200 ppm (ref. 88). However, Aroclor 1258 causes mortality at dosages above 100 ppm and partial mortalitites at dosages above 30 ppm when fed in a diet for 4 to 5 weeks. One hundred percent mortality occurs in chickens fed Aroclor 1254 above 250 ppm. Pathologically, clinical observations demonstrate that general edema occurs at all toxic dosages (refs. 7, 10, 35, 43, 55, 56). Internal haemorrhaging and tubular dilatation in kidneys are accompanied by enlargement of the kidneys, in addition to defeathering and dermatitis (refs. 86,88). Depression and secondary sexual characteristics occurred at levels as low as 30 ppm of Aroclor 1248 (ref. 7).

Sublethal effects caused by enzyme induction and increased steroid metabolism have been demonstrated in pigeons. American kestrels fed Aroclor 1254 and 1262 at levels of 0.5 and 5 ppm for 5 months affected hormone metabolism (refs. 53, 66, 67). In an interaction of polychlorinated biphenyls and duck hepatitis virus, 10-day-old ducklings fed Aroclor 1254 at levels of 25, 50 and 100 ppm had significantly higher mortality than those birds not exposed to PCB (ref. 13). Vos and Koeman (1970) found direct effects on the lymphoid system in chickens (ref. 88). Peakall and Lincer (1972) observed embryonic mortality and chromosomal aberrations in ring doves fed Aroclor 1254 at 10 ppm over two generations (ref. 54). They found that 13 of 17 PCB-treated embryos had aberration rates exceeding the mean control. Results thus indicate possible clastogenic (i.e., chromosome-breaking) action of PCB in dove embryos. Eggshell thinning was not observed in either the first or second generation. Dahlgren et al. observed that pheasant fertility and eggshell thickness were not affected by PCB exposure (ref. 10). The chief effects on reproduction occurred through PCB taken in by the hen but not by the cock. In laying pheasant hens, the major effects were observed in egg production, hatchability, and viability of the embryo. There also was a subtle effect on behavior, as the visual cliff and ability of offspring to avoid hand capture was adversely affected.

Among the most profound demonstrations of the adverse effects of PCB was the inhibition of mink reproduction that was caused by feeding them coho salmon containing PCB residues (refs. 2, 62, 63).

CONCLUSIONS

Polychlorinated biphenyls are highly persistent environmental contaminants that bioaccumulate in food chains and produce direct acute and chronic effects as well as subtle impact on the growth, reproduction, behavior, and health of fish, birds, and mammals. Freshwater fish are particularly susceptible to chronic effects since PCB residues concentrate at high levels that are known to elicit serious toxicological effects. Fish-eating birds and mammals are thus subject to hazardous levels of residues. However, the actual mode of action and ecological effects are poorly understood, as is the long-term impact of PCB on animal populations.

ACKNOWLEDGMENTS

The staff of the Fish-Pesticide Research Laboratory under the direction of Dr. Richard A. Schoettger contributed the major portion of the information on effects of PCB on aquatic organisms. The detailed *Quarterly and Annual Reports of the Research Divisions of the Bureau of Sport Fisheries and Wildlife* during 1969-1972 were especially helpful in addition to the publications and unpublished manuscripts from which the tables and figures were developed. The technical assistance of Drs. David L. Stalling, Paul M. Mehrle, Foster L. Mayer, and Mr. Jerry R. Longcore and Mrs. Laura Crane was particularly helpful.

SCIENTIFIC NAMES

Cutthroat trout (*Salmo clarki*); rainbow trout (*Salmo gairdneri*); Atlantic salmon (*Salmo salar*); coho salmon (*Oncorhynchus kisutch*); lake trout (*Salvelinus namaycush*); Japanese or coturnix quail (*Coturnix coturnin*); bald eagle (*Haliaeetus leucocephalus*); double-crested cormorant (*Phalacrocorax auritus*); caspian tern (*Hydroprogne caspia*); Fosters tern (*Sterna fosteri*); osprey (*Pandion haliaetus*); mallard duck (*Anus platyrhynchos*); great blue herons (*Ardeo herodias*); red breasted merganser (*Mergus serratus*); common merganser (*Mergus merganser*); American kestrel (*Falco sparverius*); ring dove (*Steptopelia risoria*); pigeon (*Columba livia*); pheasant (*Phasianus colchicus*); squirrel monkey (*Saimiri* *sciureus*); cynomolgus monkey (*Macaca sp.*); mink (*Mustela vison*).

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PCB's: EFFECTS ON AND ACCUMULATION BY ESTUARINE ORGANISMS

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Abstract

Effects of PCB's on and accumulation by estuarine organisms were studied in laboratory bioassays. Aroclors 1016, 1242, and 1254 were acutely toxic to certain estuarine organisms at concentrations greater than 10 μg/l, but these bioassays underestimated toxicities of PCB's, as shown by data from exposures that lasted longer than 2 weeks. Concentrations that were lethal to selected invertebrates and fishes in chronic exposures ranged from 0.1 to 5 μg/l. Reproduction of sheepshead minnows was impaired by concentrations of Aroclor 1254 in their eggs >5 μg/l, but this was not observed in eggs that contained up to 77 μg/g of Aroclor 1016. Bioaccumulation of PCB's in estuarine organisms generally exceeded 10^4 times the concentration in water in laboratory studies and 10^5 times in the estuary.

Following the discovery of PCB's in Escambia Bay, Florida, in 1969 (ref. 1), the Environmental Research Laboratory at Gulf Breeze, Florida, began studies with PCB's to determine their effects on and bioaccumulation by estuarine organisms. Estuarine organisms studied included bacteria (ref. 2); protozoans (ref. 3,4); oysters (refs. 5,6); shrimp (refs. 7,8,9,10,12,13); fishes (refs. 14,15,16,17,18,19); and communities of benthic organisms (ref. 20). The following discussion is confined to research on Aroclor® 1016, 1242, 1254 because these PCB's are presently produced in the greatest quantities.

Aroclors 1016, 1242, and 1254 are acutely toxic to certain estuarine organisms. The 48- or 96-hour LC50's, in μg/l, range from 9 to 32 for penaeid shrimp, 12 to 16 for grass shrimp, >100 for pinfish and EC50 (reduction of shell growth for oysters) from 10 to 32 μg/l (refs. 1,15,21).

Laboratory bioassays lasting longer than 2 weeks demonstrate that acute bioassays underestimate the toxicities of Aroclors 1016 and 1254. Aroclor 1254 is toxic to commercially valuable shrimps (*Penaeus* spp.) and grass shrimp (*Palaemonetes pugio*) at concentrations of 1 μg/l (refs. 8,11). Exposed shrimp are particularly sensitive to salinity stress (ref. 11) and possibly to viral disease (ref. 13). Aroclor 1254 decreases growth rates of oysters (*Crassostrea virginica*), at 4 μg/l (ref. 5). At concentrations of about 5 μg/l, Aroclor 1254 is lethal to juvenile fishes: spot (*Leiostomus xanthurus*), pinfish (*Lagodon rhomboides)*, and sheepshead minnow (*Cyprinodon variegatus*) (refs. 14,19). At Aroclor 1254 concentrations of 0.1 μg/l, some sheepshead minnow fry die (ref. 19). Aroclor 1016, at 15 μg/l, was lethal to pinfish and sheepshead minnows; susceptibilities of fry, juvenile, and adult sheepshead minnows were similar (refs. 15,18).

Aroclor 1254 affects reproduction of the sheepshead minnow, an estuarine fish (ref. 16). Adult fish exposed for 4 weeks to 0.1 μg/l appeared to be unaffected, but when eggs from these fish were fertilized and placed in PCB-free water, the survival of fry was diminished. Mortality was observed also in fry from eggs that contained greater than 5 μg/g of the PCB and increased as PCB content of the eggs increased. Reproductive success of Atlantic salmon (ref. 22) and striped bass (ref. 23) may also be affected by PCB's in their eggs. Adult sheepshead minnows that had been exposed for 4 weeks to from 0.3 to 3 μg/l of Aroclor 1016 produced eggs that contained from 3 to 77 μg/g of this PCB, yet fry that hatched from these eggs were apparently unaffected (ref. 18).

Estuarine organisms accumulate PCB's from water. Maximum concentration factors (concentration in animals divided by concentration in water) in laboratory exposures of various species to Aroclor 1254 were: oysters, 101,000 (ref. 5) to 165,000 (ref. 6); shrimp, 26,000 (ref. 9); and fishes, 37,000 (refs. 14,19). The maximum concentration factor determined from exposures of fishes to Aroclor 1016 was 34,000 (refs. 15,17). Fishes exposed continuously to Aroclors 1016 and 1254 accumulated both Aroclors in increasing concentrations for about 4 weeks; thereafter, the quantity stabilized (refs. 14,15). Maximum concentrations were found in the liver, gills, and skin of the fish, but the lowest concentration found in fishes and shrimps was in the muscle (refs. 14,15,8). Bioconcentration factors calculated from data from Escambia Bay (refs. 1,10) were greater than 230,000 for shrimp, 670,000 for fishes, and greater than 100,000 for oysters, indicating that laboratory data can underestimate bioconcentration potentials of Aroclor 1254.

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SUMMARY OF RECENT INFORMATION REGARDING EFFECTS OF PCB'S ON FRESHWATER ORGANISMS

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Abstract

Polychlorinated biphenyls (PCB's) are toxic to freshwater organisms at concentrations below 5 μg/l (ppb), and newly hatched fish and small insects and crustaceans with short life cycles are most sensitive. Aroclor 1254 at 0.45 μg/l produced a 50 percent decrease in midge reproduction; 1.3 μg/l caused a 50 percent reduction in Daphnia reproduction; and 1.8 μg/l produced 50 percent reduction in fathead minnows. The indirect toxicity of PCB's to predators through accumulation of PCB's in tissues of food organisms causes death. Fish can accumulate 200,000 times more PCB's in their flesh than can be held in the surrounding water. The concentration of Aroclor 1260 in the water resulting in a tissue residue of 0.5 μg/l is approximately 0.002 μg/l. Aroclor 1016 and 1242 have been shown to have similar toxicity to freshwater organisms.

The Environmental Protection Agency proposed water quality criterion for PCB's is as follows: "Maximum acceptable concentration of PCB's in water is 0.001 μg/l."

Polychlorinated biphenyls (PCB's) are toxic to freshwater fish and other aquatic organisms at very low levels and direct toxic effects are evident within a few hours, but much greater harm is caused by longer exposure at lower concentrations. PCB levels that kill fish, crustacea, and aquatic insects are similar to those of DDT and several other pesticides. Environmental occurrence of these materials is increasing and there is mounting evidence that they are causing widespread harm in the freshwater aquatic environment.

One of the significant accomplishments since the discovery of the toxicity of PCB's to freshwater organisms was the synthesis of known data and their publication in the "Blue Book," or *Water Quality Criteria—1972*, sponsored by the U.S. Environmental Protection Agency and prepared by the National Academy of Sciences, National Academy of Engineering Committee on Water Quality Criteria (ref. 1). It was recommended in this report that aquatic life should be protected where the maximum concentration of total PCB in unfiltered water does not exceed 0.002 μg/l at any time or place, and the residues in the general body tissues of any aquatic organism do not exceed 0.5 μg/g.

The Environmental Protection Agency (ref. 2) also proposed water quality criteria for PCB's as follows: "Maximum acceptable concentrations of PCB's in water are 0.002 μg/l, and maximum acceptable levels of residues in general body tissues of any aquatic organism are 0.5 μg/l"; following the proposed criteria recommended by the National Academy "Blue Book." However, these criteria have not been promulgated. Recently, the Environmental Protection Agency (ref. 3), using the latest information available, has proposed 0.001 μg/l PCB as a maximum permissible concentration in freshwater, but has proposed no tissue level criterion.

The purpose of this paper is to present a summary of recent information dealing with the toxicity of polychlorinated biphenyl compounds to freshwater fish, crustaceans, and aquatic insects, and to present important recent information on the bioconcentration of PCB's in fish tissues and its effect on consumer organisms.

Fish

A significant amount of new information dealing with the toxicity of PCB's to fish has been collected during the last 3 years. Acute and longer term studies have been conducted with a variety of freshwater sport and forage fish species. Tests with fathead minnows at the National Water Quality Laboratory, Duluth, Minnesota, (ref. 4) have shown that Aroclor 1242, A-1248, and A-1254 are toxic below 5 μg/l. Ninety-six-hour LC_{50} values (that concentration which kills half the fish in 96 hours) for newly hatched fathead minnows were 15 μg/l for Aroclor 1242 and 7.7 μg/l for A-1254. Fifty percent died at 8.7 μg/l A-1248 after 30 days. After 60 days, half were dead at 8.8 μg/l A-1242 and 4.6 μg/l A-1254 (table 1).

Two 9-month continuous-flow bioassay tests were conducted (ref. 4) with the fathead minnow to determine the effects of Aroclor 1242 and A-1254 on survival and reproduction (table 2). Concentrations of Aroclor 1242 above 10 μg/l were lethal to newly hatched fry and all minnows were dead after 96 hours in 51 μg/l A-1242; none survived to the end of the test at 15 μg/l. Fry survived when reared in the same concentrations at which their parents lived and spawned; however growth of newly hatched fish was retarded prior to their death

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Table 1. Acute toxicity of Aroclor 1242, 1248, and 1254 to fathead minnows, Gammarus and Daphnia, in continous-flow tests

	Test temp. (C)	96-hr LC_{50}[a] (µg/l)			60-day LC_{50} (µg/l)		
		1242	1248	1254	1242	1248	1254
Fathead minnow, newly hatched	24°	15	*[b]	7.7	8.8	8.7[c]	4.6
Gammarus	18°	73	29	--	8.7[c]	5.1[c]	--
Daphnia	18°	--	12.9	6.4	--	2.6[d]	1.8[d]

[a] Concentration where 50 percent died after 96 hours.

[b] No mortality after 96 hours.

[c] Thirty-day LC_{50}.

[d] Fourteen-day LC_{50}.

Table 2. Spawning and egg production of fathead minnows exposed to Aroclor 1242 and 1254

Aroclor concentration (µg/l)	Number of spawnings per female	Number of eggs per spawning	Number of eggs per female	Percent eggs hatched
Aroclor 1242				
51.0	0	0	0	0
15.0	0	0	0	0
5.4	2.5	30	151	81
2.9	3.9	63	283	38
0.9	1.3	28	35	84
0.0	4.6	90	442	53
Aroclor 1254				
15.0	0	0	0	0
4.6	0	0	0	0
1.8	1.0	63	106	79
0.5	5.2	105	556	63
0.2	3.0	64	221	55
0.0	2.4	104	253	75

larvae of the mosquito *Culex tarsalis*, exposed to 1.5 μg/l Aroclor 1254, survived well and pupated, but many of the pupae were unable to metamorphose into the adult form. Control organisms pupated with success.

Bioconcentration

Bioconcentration, biological magnification, bioaccumulation, PCB or tissue residues, and residue dynamics, are some of the terms used to describe the intake of PCB's from food or direct water contact into the animal and accumulation in the tissues to concentrations many thousands of times those in the water or food they were exposed to. Polychlorinated biphenyls are much more soluble in fatty tissues (lipids) than water and are selectively concentrated in those tissues. Animals that feed on aquatic invertebrates or fish that have bioaccumulated significant tissue concentrations of PCB's are threatened even when the prey species are not adversely affected.

Exposure of fish to water containing PCB's demonstrates that they can bioconcentrate PCB's directly from the water, in addition to uptake in food, and in most cases direct uptake from water is more rapid and leads to much higher accumulation in the tissues. The bioconcentration factor (concentration in fish tissue/concentration in the water) for PCB's is essentially independent of the PCB concentration in the water. The bioconcentration factor (whole body) for adult male fathead minnows at 25° C is approximately 1.2×10^5 (120,000) for Aroclor 1248 and 2.7×10^5 (270,000) for Aroclor 1260. Female fathead minnows accumulated about twice as much PCB's as the male fatheads and the difference is largely

The residue of PCB's in the tissues of fathead minnows studied by DeFoe et al. (ref. 7) was directly proportional to the concentration of the toxicant in the water. The concentration of Aroclor 1248 in the water resulting in a tissue residue of 0.5 μg/g was approximately 0.004 μg/l. The concentration of Aroclor 1260 in the water resulting in a tissue residue of 0.5 μg/g was approximately 0.002 μg/l. They showed that the more highly chlorinated PCB mixtures are bioconcentrated to a greater extent in the lipids of the fish than the lower chlorinated compounds. They also showed that the PCB residues in the fish reached an apparent steady state after approximately 100 days, and when exposed fish were placed in untreated Lake Superior water they eliminated less than 18 percent of the body burden after 60 days.

Nebeker et al. (ref. 4) showed that the bioconcentration factor for Aroclor 1254 in fathead minnows ranged from 1.1×10^5 to 2.4×10^5 and that the bioconcentration factor for several PCB's in the fathead minnow ranged from about 0.3×10^5 to 2.7×10^5 with the more highly chlorinated PCB's accumulating to a greater extent. Adult fathead minnows accumulated up to 430 ppm A-1242 in whole body residues, with concentrations up to 270,000 times that of water, and up to 1,000 ppm of 1254, with concentrations up to 230,000 times that of the water (ref. 4), *Gammarus* accumulated over 500 ppm 1248 and 400 ppm 1242, with concentrations over 100,000 times that of the water (ref. 14).

Sodergren and Svensson (ref. 19) showed that the nymph of *Ephemera danica* accumulated Clophen A 50 to bioconcentration factors of 3×10^3. After 60 days of exposure to Aroclor 1242 and A-1248, the maximum bioconcentration factor in *Gammarus pseudolimnaeus* was approximately 3×10^4 and 5×10^4, respectively (ref. 14).

Sanders and Chandler (ref. 18) showed that the uptake and biological magnification of Aroclor 1254 by some aquatic invertebrates was very rapid. When *Daphnia magna* were exposed for 4 days to water containing 1.1 μg/l of A-1254, they accumulated total body concentrations 48,000 (4.8×10^4) times greater than those in water. The mosquito larvae *Culex tarsalis* exposed to water containing 1.5 μg/l A-1254 accumulated 19 μg/g of this compound within 24 hours, which represents a 12,600-fold magnification. Scuds, *Gammarus pseudolimnaeus*, exposed continuously to 1.6 μg/l A-1254, reached an equilibrium concentration in 14 days. Total body residues were 44 μg/g, a concentration 27,500 times that in the water.

Some evidence exists that the bioconcentration factors measured in the laboratory are essentially the same as those found in the river environment. Veith and Lee (refs. 20, 21) reported that goldfish in the lower Milwaukee River accumulated Aroclor 1248 approximately 0.7×10^5 to 2×10^5 times (depending on the lipid content) the estimated PCB concentration in the river water. The maximum concentration of Aroclor 1254 in Saginaw River shad was 165 μg/g whereas typical water concentrations averaged 1.1 μg/l, with a maximum of 2.9 μg/l (ref. 22). This indicates a bioconcentration of 0.6×10^5 to 1.5×10^5.

Studies resulting from the reproductive failure of mink fed Great Lakes fish led to the conclusion that it was PCB residues in the fish that caused the adverse effects. Ringer et al. (ref. 23) demonstrated that 10 μg/g of Aroclor 1254 in coho salmon produced 71 percent mortality in mink and that a mixture of 10 μg/g PCB's and 0.5 μg/g dieldrin in coho feed produced 100 percent mortality. No kits were born alive when the diet contained 5 μg/g or greater Aroclor alone. The "no effect" concentration of PCB's in the mink diet was estimated to be less than 1.0 μg/g.

Platonow and Karstad (ref. 24) presented similar at higher PCB concentrations. Reproduction occurred and good egg hatching took place at and below 5.4 μg/l A-1242. Eggs produced by control fish but maintained at the higher concentrations of 15 and 51 μg/l hatched with good success but none of the fry survived.

Aroclor 1254 was more toxic to the fathead minnow than A-1242 (ref. 4), as they did not survive and reproduce above 1.8 μg/l A-1254 (table 2). All fish in the long-term study were dead after 96 hours at 15 μg/l, and growth was delayed at 4.6 μg/l. Spawning occurred at 1.8 μg/l but was significantly less than at the lower concentrations. Egg hatchability and fry survival were good at and below 1.8 μg/l. Eggs produced by the control fish and maintained at 15 μg/l hatched readily, but all young fish were dead within 96 hours.

Growth of young fathead minnows was affected above 2.2 μg/l A-1248, and none survived above 5.1 μg/l after 30 days. Young flagfish did not survive at Aroclor 1248 concentrations above 5.1 μg/l and did not grow well above 2.2 μg/l (ref. 4).

Due to low PCB solubility, 96-hour LC_{50} values do not adequately reflect PCB toxicities to fish. For Aroclor 1221-1268, 96-hour LC_{50} values ranged from 1,170 to 50,000 μg/l for cutthroat trout. The acute oral toxicity of Aroclors 1242, 1248, 1254, and 1260 was greater than 1,500 mg/kg in rainbow trout. Intermittent-flow bioassays of A-1242, 1248, and 1254 to bluegills resulted in 15-day LC_{50} values of 54, 76, and 204 μg/l, respectively. Exposures to channel catfish gave 15-day LC_{50} values of 107, 127, and 741 μg/l for the same Aroclors. All LC_{50} values decreased significantly with longer exposure, indicating the much greater PCB hazard with increased exposure time (ref. 5). Direct water exposures appear to represent a greater hazard to fish than dietary exposures, but dietary sources are important as PCB's have a high affinity for sediments and readily enter the food chain.

Schoettger et al. (ref. 6) showed that the growth of lake trout was retarded by Aroclor 1248 in the diet for 3 months. Concentrations of 1.2, 3.8, and 12 μg/g decreased weight gain by 6, 10, and 28 percent, respectively, below that of controls. The growth of the high-exposure group (12 μg/g in diet) was only about half that of the controls after 6 months.

Fathead minnows were exposed to Aroclor 1248 and A-1260 (ref. 7), in flow-through bioassays to determine the acute (30-day) and chronic (240-day life cycle) effects on the fry and adult fathead minnows. Newly hatched young were the most sensitive lifestage to the exposure of PCB's. Total mortality occurred when newly hatched fatheads were exposed for 30 days to 8.5 μg/l A-1248. All fish exposed to 9.0 μg/1 A-1260 died after a 30-day exposure. The calculated 30-day TL_{50} for newly hatched fathead minnows (<8 hours old) was 4.7 μg/l for A-1248 and 3.3 μg/l for Aroclor 1260. PCB's did not measurably affect the ability of fathead minnows to reproduce at concentrations which were acutely toxic to the larvae. With reproduction occurring at and below 3 μg/l for A-1248 and at below 2.1 μg/l for A-1260, the 20 percent reduction in the standing crop in the second generation of minnows at concentrations as low as 0.4 μg/l is due to the mortality of the fathead larvae soon after hatching.

Aroclor 1016, a compound chemically similar to A-1242, has been introduced recently to replace PCB's of the Aroclor 1200 series in many applications. Work has been completed with this compound with freshwater organisms and what is available indicates that the toxicity of Aroclor 1016 is similar to that of Aroclor 1242.

Mayer (ref. 8) presents data on the toxicity of Aroclor 1016 to nine species of freshwater fishes. Bluegills had a 4-day LC_{50} of 260 μg/l A-1016; rainbow trout, 135 μg/l; Atlantic salmon, 134 μg/l; and yellow perch, 185 μg/l A-1016.

Johnson (ref. 9) presented data comparing the effects of A-1242 and A-1016 on rainbow trout and bluegill sunfish. Rainbow trout had a 10-day LC_{50} of 39 μg/l for A-1242 and 17-day LC_{50} of 49 μg/l for A-1016. Bluegills had a 15-day LC_{50} of 54 μg/l A-1242 and a 35-day LC_{50} of 43 μg/l for A-1016. They had 96-hour LC_{50} values of 125 μg/l A-1242 and 46 μg/l A-1016.

A test comparing the toxicity of Aroclor 1242 and Aroclor 1016 using fathead minnows was completed by Veith (ref. 10). There was no difference in the survival of fry after 96 hours between A-1242 and A-1016 at 28 μg/l; both had 50 percent mortality. In 30-day tests, 100 percent mortality of fry occurred at 42 μg/l A-1242 and 95 percent mortality occurred at 42 μg/l A-1016. Approximately 50 percent had died after 30 days at 25.5 μg/l A-1242 and 30.5 μg/l A-1016.

The effects of Aroclor 1242 on the guppy *Poecilia reticulata* were studied by Morgan (ref. 11) in static tests. The guppy had 100 percent mortality at 20 and 2 ppm, but only 25 percent mortality at 0.2 ppm after 7 days.

In preliminary investigations, Jensen, et al. (ref. 12) reported a possible relationship between PCB residues in salmon eggs and egg mortality in Sweden. When residues in groups of eggs ranged from 0.4 to 1.9 μg/g on a whole-weight basis (7.7 to 34 μg/g on a fat basis), related mortalities ranged from 16 to 100 percent.

No adverse effects were observed on survival, growth, and reproduction of brook trout exposed for 71 weeks to 0.94 μg/1 and lower concentrations of the PCB

Aroclor 1254. Survival and growth to 90 days of alevin-juveniles from exposed parents were also unaffected. PCB concentrations in the brook trout were directly proportional to the water exposure concentration. PCB tissue concentrations appeared to have reached a steady state by the first sampling following 14 weeks of exposure. PCB residues (wet tissue basis) in chronically exposed fish were approximately 2 μg/g in the fillet and 9 μg/g in the "whole body" (minus one fillet and the gonads) at the highest water concentration, 0.94 μg/1. The higher residue in the "whole body" compared to the corresponding fillet was due to the higher fat content of the former (ref. 13).

Thyroid stimulation in coho salmon ranged from a 52 percent increase in fish fed 0.48 μg/g Aroclor 1254 of food to a 119 percent increase in the high-treatment group (480 μg/g). The lowest detectable stimulation of thyroid activity occurred in salmon dosed with 1/1000th that caused mortality. In channel catfish, 2.4 μg/g and 24 μg/g A-1254 in food caused significant increases in thyroid activity when compared to control fish (ref 15).

Crustacea

Freshwater crustaceans are very sensitive to low concentrations of PCB's. Because of their generally short life cycles they will be eliminated from PCB-contaminated waters quite rapidly, causing not only their loss from aquatic systems but impairment of fish populations because of a decrease in fish food organisms.

To assess the danger of PCB compounds to fish food organisms, bioassays were conducted with *Daphnia magna* (ref. 14) using the eight commercially available PCB's, Aroclor 1221, 1232, 1242, 1248, 1254, 1260, 1262, and 1268 (table 3). Aroclor 1248 was the most toxic to *Daphnia magna* of the eight Aroclors tested in static tests; the 3-week LC_{50} was 25 μg/l. Aroclor 1232, 1242, and 1254 had similar toxicity values to A-1248. In continuous-flow Daphnia tests, Aroclor 1254 was the most toxic, with a 3-week LC_{50} of 1.3 μg/l. No reproduction occurred at 3.8 μg/l A-1254, and no adults survived 3.5 μg/l. The Aroclors were much more toxic in continuous-flow conditions due to continual replacement of fresh PCB. Almost one-third of the PCB added to the test water was lost to the air, with some lost to bacteria, algae, waste materials, test container surfaces, and test animal tissues. The difference between static and continuous-flow results (table 3) illustrates how critical test methodology can be for certain chemicals.

Table 3. Toxicity of eight Aroclors to *Daphnia magna* in static and continous flow bioassays

Aroclor (PCB)	3-week LC_{50}[a] (μg/l)	50 percent reproductive[b] impairment (μg/l)
Continuous-flow test conditions		
1248	2.6	2.1
1254	1.8[c]	1.1
1254	1.3	1.3
Static test conditions		
1221	180	125
1232	72	66
1242	67	63
1248	25	24
1254	31	28
1260	36	33
1262	43	41
1268	253	206

[a] Concentration where 50 percent died.

[b] Fifty percent loss of young.

[c] Two-week LC_{50}.

The freshwater scud *Gammarus pseudolimnaeus* was also studied by Nebeker and Puglisi (ref. 14) in continuous-flow test systems to determine the effects of A-1242 and A-1248 on survival and reproduction. Ninety-six-hour LC_{50} values were 73 μg/l for A-1242 and 29 μg/l for A-1248. Fifty percent were surviving after 60 days at 8.7 μg/l A-1242 and 5.1 μg/l A-1248. No live animals remained after 2 months at 26 μg/l A-1242 or 18 μg/l A-1248 (table 4). Good reproduction occurred at and below 2.8 μg/l A-1242 and 2.2 μg/l A-1248.

Static and flow-through toxicity tests with aquatic invertebrates (ref. 5) indicate that they are generally more susceptible to acute toxic effects of PCB's than fish. *Gammarus faciatus* in flow-through tests had a 96-hour LC_{50} of 10 μg/l A-1242 and a 10-day LC_{50} of 5 μg/l A-1242. The glass shrimp *Palaemonetes kadiakensis* was very sensitive to A-1254, with a 7 day LC_{50} of 3 μg/l.

In static tests with the amphipod *Gammarus pseudolimnaeus*, Mayer et al. (ref. 15) found that of four PCB homologs tested, the trichlorobiphenyl was slightly more toxic than the di-, penta-, and hexachlorobiphenyls. The trichlorobiphenyl had a 96-hour LC_{50} of 100 μg/l, two dichlorobiphenyls tested had 96-hour LC_{50} values of 100 and 120 μg/l, the hexa- and penta-homologs had 96-hour LC_{50} values of 150 and 210 μg/l respectively.

The crayfish *Orconectes nais* was reported by Stalling and Mayer (ref. 5) to have a 7-day LC_{50} of 30 μg/l for Aroclor 1242 and a 7-day LC_{50} of 80 μg/l for A-1254.

Table 4. Survival and reproduction of *Gammarus pseudolimnaeus* after 2 months exposure to Aroclor 1242 and 1248

Aroclor concentration (µg/l)	Survival of adults (percent)	Young per surviving adult
Aroclor 1242		
234.0	0	0
81.0	0	0
26.0	0	0
8.7	52	0
2.8	77	4
0.0	48	7
Aroclor 1248		
18.0	0	0
5.1	53	7
2.2	73	22
0.5	71	20
0.2	73	11
0.0	64	11

Daphnia pulex were quite sensitive to Aroclor 1242 (ref. 11). Young *Daphnia pulex* died at levels as low as 0.02 ppm in 4-day static tests. The toxicity of Aroclor 1242 and DDT to the ostracod *Cypidopsis vidua* appeared to be approximately the same in static tests. At 20 ppm, there were no survivors out of 20 ostracods in the Aroclor cultures, and some mortality occurred at 2 ppm.

Studies by Moorman and Biesinger (ref. 16) comparing the lethality of Aroclor 1016 and Aroclor 1242 to *Daphnia magna* have shown similar toxicity for both compounds in static tests. No young survived after 3 weeks at 125 µg/l A-1016 or A-1242. At 100 µg/l, 80 percent of the young (percent of control) survived after 3 weeks in A-1016 and 19 percent of the young survived A-1242. Good survival occurred at 50 µg/l for both compounds.

A study of the toxicity of isomers of Aroclor 1242 and 1254 to *Daphnia magna* by Biesinger (ref. 17) showed significant reproductive impairment in static tests at 50 µg/l for trichloro- and tetrachlorobiphenyls, indicating that there was little difference in the toxicity of the tri- and tetra- chlorobiphenyl compounds.

Aquatic Insects

Aquatic insects also appear to be very sensitive to longer exposures of PCB's, though response is variable when exposed to brief, or acute, concentrations. The midge *Tanytarsus dissimilis* was tested through its full life cycle (ref. 14) and found to be affected at levels below 1 µg/l. Survival and growth of the midge, when tested with Aroclor 1254, were excellent in control chambers but were reduced by 50 percent at the lowest test concentration of 0.45 µg/l (table 5). At 1.2 µg/l, the numbers of larval cases were reduced to 35 percent of the control, and the numbers of pupal cases were reduced to 18 percent of the control. No adult emergence occurred above 3.5 µg/l, and abundant adult emergence did not occur above 3 µg/l, even though larvae were present. No pupal cases were constructed at 9 µg/l and no larval cases were formed at 33 µg/l A-1254. The calculated 3-week LC_{50} for A-1254 (50-percent reduction based on control as 100 percent) was 0.65 µg/l for larvae and 0.45 µg/l for pupae. Adult midges emerged at concentrations up to 9 µg/l Aroclor 1248. Larvae were present at 18 µg/l, but adult emergence did not occur. Abundant emergence did not occur above 5.1 µg/l (ref. 12).

Mayer et al. (ref. 15) report some acute data for other aquatic insects. Dragonfly and damselfly nymphs, *Macromia sp.* and *Ischnura verticalis*, were tolerant to Aroclor 1242 and A-1254 in short-term static tests. The dragonfly had a 7-day LC_{50} of 800 µg/l for Aroclor 1242 and 1,000 µg/l for A-1254. The damselfly, tested under continuous-flow conditions, had a 4-day LC_{50} of 400 µg/l for A-1242 and 200 µg/l for A-1254.

Studies by Sanders and Chandler (ref. 18) have shown that larvae of the stonefly *Pteronarcys dorsata*, the dobonfly *Corydalus cornutus*, and the phantom midge *Chaoborus punctipennis* survived concentrations of up to 2.8 µg/l A-1254 for up to 21 days without significant mortality. However, in similar experiments,

Table 5. Effect of Aroclor 1254 on the growth and survival of the midge *Tanytarsus dissimilis*

Aroclor concentration (µg/l)	Number of mature larval cases (percent of control)	Number of pupal cases (percent of control)
33.0	0	0
9.0	0.2	0
3.5	7	7
1.2	35	18
0.4	52	55
0.0 (control)	100	100

evidence for mink fed diets enriched with Aroclor 1254. The data show that 0.64 μg/g of PCB in the rations resulted in only 1 of 12 mink producing 3 kits, all of which died on the first day.

Research with nonhuman primates indicates that the toxicity of PCB residues to consumers are not limited to mink. Barsotti and Allen (ref. 25) and Allen et al. (ref. 26) have shown that PCB enriched diets were toxic to primates. Female primates fed 2.5 μg/g Aroclor 1254 in their diet had periorbital edema, acneform lesions on face and neck, and substantially reduced fertility after 2 months.

SUMMARY

There is ample evidence that PCB's are toxic to freshwater organisms at very low concentrations. Newly hatched fish and small insects and crustaceans with short life cycles appear to be the most sensitive life forms. In larger animals or those with longer life cycles, toxic effects are delayed and acute or short-term testing does not adequately reflect the chronic or longer term effects that PCB's have on the test animals. Concentrations of polychlorinated biphenyls commonly found in contaminated waterways today, such as Aroclor 1242 and 1254, are directly toxic to fish and aquatic invertebrates in the range of 0.1 to 10.0 μg/l. Aroclor 1016 and 1242 have been shown to have similar toxicity to freshwater organisms. The indirect toxicity of PCB's to predators through accumulation of PCB's in tissues of food organisms may cause deaths from water concentrations that do not cause direct lethality.

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DISTRIBUTION AND EXCRETION OF [^{14}C]-2,4,5,2′,5′-PENTACHLOROBIPHENYL IN THE LOBSTER (*Homarus americanus*) AND THE DOGFISH SHARK (*Squalus acanthias*)*

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Abstract

Significant ambient concentrations of polychlorinated biphenyl (PCB) residues were present in lobster hepatopancreas (1.57 ppm wet weight) and dogfish shark liver (1.87 ppm wet weight). PCB concentrations in muscle were only 10-20 percent of hepatic values. After injection of [^{14}C]-2,4,5,2′,5′-pentachlorobiphenyl (^{14}C-2,4,5,2′,5′-PCB) (0.2 mg/kg) into the pericardial sinus of the lobster, the hepatopancreas contained more than 90 percent of the recovered radioactivity at all times investigated (24 hr-8 wk). Virtually all of the hepatopancreas radioactivity was unaltered 2,4,5,2′,5′-PCB. The radioactivity was very persistent in hepatopancreas, female egg mass, stomach, gill, and intestine of lobsters ($t_{1/2} > 3$ wk). Following intravascular administration of ^{14}C-2,4,5,2′,5′-PCB (0.03 mg/kg) to dogfish, almost all of the dosed radioactivity was found in liver at each time studied (6 hr-12 days). Most of the benzene extractable material was also unaltered 2,4,5,2′,5′-PCB in each instance. Small amounts of radioactivity were excreted in bile but only traces in urine. Microsomes from lobster hepatopancreas had no detectable aniline hydroxylase, benzphetamine demethylase, 7-ethoxycoumarin O-deethylase, or benzpyrene hydroxylase activity although dogfish shark liver microsomes had low activity with each substrate tested. The persistence of ^{14}C-2,4,5,2′,5′-PCB in the two marine species is probably related to the low hepatic microsomal mixed-function oxidase activities observed and the high lipid content of lobster hepatopancreas and dogfish shark liver.

INTRODUCTION

The polychlorinated biphenyls (PCB's) possess high chemical and thermal stabilities and have been used extensively in the United States for certain industrial applications during the past 45 years (ref. 1). PCB's were first recognized as environmental contaminants in 1966 (ref. 2). Subsequently, the widespread occurrence of PCB residues was verified in wildlife species (ref. 3) (both terrestrial and marine) all over the world, in human adipose tissue (ref. 4), in waters of the North Atlantic (ref. 5), and in marine species indigenous to Maine (ref. 6). The chemical stability and lipid solubility of certain PCB's contributes to their bioaccumulation.

Commercially available PCB's are complex mixtures of many different species with varying chlorine content. Aroclor 1254, for example, was resolved into more than 50 individual compounds, of which the pentachlorobiphenyl derivatives were the major constituents (ref. 7).

The present study describes the distribution and excretion of [^{14}C]-2,4,5,2′,5′-pentachlorobiphenyl (^{14}C-2,4,5,2′,5′-PCB) in the lobster (*Homarus americanus*) and the dogfish shark (*Squalus acanthias*) after parenteral administration. The in vivo data is correlated with in vitro microsomal mixed-function oxidase activities of lobster hepatopancreas and dogfish shark liver. A single, radiolabeled PCB isomer was used in the disposition experiments. Use of pure isomers has obvious advantages over similar experiments with commercial PCB mixtures. The isomer chosen is a constituent of Aroclor 1254, and should be representative of several Aroclor 1254 components. PCB residues were also determined in certain tissues of lobsters and dogfish sharks.

MATERIALS AND METHODS

Male or female lobsters (320-560 g) were purchased locally (Mt. Desert Island, Maine) and female dogfish sharks (3.0-5.1 kg) were caught by hook in Frenchman Bay, Maine. Both species were maintained in tanks equipped with circulating fresh seawater before and after treatment with ^{14}C-2,4,5,2′,5′-PCB. The lobsters maintained for longer than 1 week after dosing were submerged in a lobster crate in Frenchman Bay. They were fed small pieces of mackerel or flounder three times a week when the traps were lifted to remove any dead or ill animals. Only lobsters that were alive and in apparent good health were dissected at the indicated times.

2,4,5,2′,5′-pentachloro [^{14}C] biphenyl (^{14}C-2,4,5,2′,5′-PCB), (10.98 mCi mmole) was obtained from Mallinckrodt Nuclear and was shown by thin-layer chromatographic analysis (TLC) to be greater than 98.5 percent radiochemically pure.

^{14}C-2,4,5,2′,5′-PCB was injected into the pericardial sinus of lobsters (0.2 mg/kg, 1.4 × 10^7 dpm/kg) or into the caudal vessels of dogfish sharks (0.03 mg/kg, 2.2 × 10^6 kpm/kg) as a solution in Emulphor water (36-38 percent v/v).

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Lobster plasma samples were withdrawn from the pericardial sinus just prior to sacrifice and placed in vials containing citrate. Dogfish blood samples (serially in disappearance studies or terminally in distribution studies) were taken from the caudal vessels and placed in heparinized vials. At various times after administration of ^{14}C-2,4,5,2',5'-PCB, animals were sacrificed and various tissues and fluids were removed from lobsters and dogfish. Duplicate aliquots of plasma, blood, bile, or cerebrospinal fluid (100 μl) or tissue (70-200 mg) were solubilized in 2 ml NCS (Amersham/Searle) by incubation at 50° C overnight. Liquid scintillation solvent (18 ml; 5.0 g PPO; and 250 mg POPOP/l toluene) was added and the radioactivity determined in a Nuclear Chicago Mark I scintillation spectrometer. Counting efficiency was determined using the channels ratio technique and varied from 50-80 percent for the solubilized biological samples. All counts were converted to disintegrations per minute.

TLC was performed on precoated silica gel GF plates (5 × 20 cm, 250 μm thick; Analtech) that were activated at 110° C for 60 min before use. Plates were developed in hexane (solvent A) or benzene:ethyl acetate, 12:1 (solvent B) and were sequentially scraped (1 cm bands). The radioactivity present in each segment was determined by counting the silica gel in vials containing 10 ml Fluoralloy (Beckman)-dioxane liquid scintillation cocktail. Radioactive recovery from the plates was normally greater than 95 percent with this procedure. Statistical analysis of the data was performed using Dunnetts' multiple comparisons test. Log transformation was used to equal the variances.

Microsomes were prepared from lobster hepatopancreas or dogfish liver homogenates (in 1.15% KCl buffered to pH 7.6 with 0.013 M HEPES) by centrifugation; microsomal protein concentrations were determined and mixed-function oxidase activities were measured toward aniline, benzphetamine, benzo[a]pyrene, and 7-ethoxycoumarin as described in detail elsewhere (ref. 8).

PCB residue analyses were performed according to Armour and Burke (ref. 9) following caustic hydrolysis of a preliminary extract which removed DDT, PCB's, and related chemicals from the tissues to be studied (ref. 10).

In estimating the degree of metabolism of ^{14}C-2,4,5,2',5'-PCB, 25 percent w/v aqueous homogenates of lobsters hepatopancreas and green gland were extracted four times with equal volumes of warm hexane and/or acidified toluene. Homogenates of livers from dogfish were extracted in a similar manner with benzene.

RESULTS

Of the various lobster tissues investigated, the female egg mass was found to contain the highest residual level of PCB's (table 1) although the hepatopancreas also contained a significant amount. This is interesting since the tomale (i.e., hepatopancreas of the cooked lobster) is considered a delicacy by some people. The PCB concentration in lobster muscle was about an order of magnitude lower than that of egg mass or hepatopancreas and was well below the 5 ppm limitation on PCB residues in human foodstuffs.

Dogfish shark liver also had appreciable PCB residues (table 1), whereas muscle contained only about one-fifth the hepatic concentration (per gram wet weight). These residual PCB's are not unexpected in either dogfish shark liver or lobster hepatopancreas since both tissues have high lipid contents (50 to 60 percent wet weight)(ref. 11).

The specific activity of several lobster tissues, plasma, and feces with time, after parenteral administration of ^{14}C-2,4,5,2',5'-PCB, is shown in table 2, and the percentage of dosed radioactivity present in the various organs at each time point is outlined in table 3. The hepatopancreas contained much higher amounts of radioactivity (per mg tissue) than any other organ (table 2) and accounted for between 91 and 96 percent of the total recovered radioactivity at all times. The half-life ($t_{1/2}$) for radioactivity in tissues was estimated from semilogarithmic plots of the specific activity (dpm/mg) versus time (days). The tissues could be classified into two categories (table 2): those in which labeled compound had a $t_{1/2}$ of 3 weeks or longer and those in which the $t_{1/2}$ was 1 week or less.

Almost all of the radioactivity in lobster hepatopancreas at 2, 4, or 8 weeks after treatment was unchanged 2,4,5,2',5'-PCB. More than 90 percent of this hepatopancreas activity was extracted into hexane after shaking homogenates four times with this solvent (93 ± 3%, mean ± SD, n = 4). Over 95 percent of the hexane soluble radioactivity cochromatographed with authentic 2,4,5,2',5'-PCB (Analabs) on TLC plates developed in solvent A (R_f 0.7-0.8) or solvent B (R_f 0.9-1.0). Similarly, more than 90 percent of the radioactivity in hexane extracts of green glands from lobsters sacrificed 2 or 4 weeks after dosing was 2,4,5,2',5'-PCB, although in this case only about 50 percent of the total activity was hexane and toluene (acidified) extractable, the remainder residing in the aqueous phase of green gland homogenate. Only about 10 percent of the fecal radioactivity was extracted into hexane and acidified toluene (four

Table 1. Residue levels of polychlorinated biphenyls (PCB's) in some tissues of the lobster *(Homarus americanus)* and the dogfish shark *(Squalus acanthias)*

Species	ppm wet weight PCB's[a]		
Lobster[b]	Egg mass	Hepatopancreas	Muscle
	4.41 (2.78-5.98)	1.57 (1.13-2.27)	0.097 (0.07-0.13)
Dogfish shark[c]	Liver	Muscle	Kidney
	1.87	0.42	0.2[d]

[a] Reported in terms of total Aroclor 1242 and 1254 residues.

[b] Mean and ranges for 4 animals.

[c] Mean values for 2 dogfish weighing 4-6 kg.

[d] Detection limit in dogfish kidney.

Table 2. Specific activity of various lobster tissues and fluids with the following pericardial injection of ^{14} C-2, 4, 5, 2^1, 5^1, -pentachlorobiphenyl (0.2 mg/kg)[a]

	Time after administration					
Tissue	24 hr	1 wk	2 wk	4 wk	8 wk	$t_{1/2}$ (days)
	dmp/mg tissue or /µl plasma					
Hepatopancreas	183.4 ± 24.7 (6)[b]	192.9 ± 33.4 (7)	170.9 ± 40.0 (5)	131.2 ± 34.7 (6)	77.4 ± 28.4 (9)	45
Green gland	20.1 ± 13.0 (6)	8.4 ± 2.4 (7)	6.1 ± 1.9 (5)	4.8 ± 1.2 (6)	3.4 ± 1.4 (9)	<7
Intestine	14.0 ± 7.0 (6)	14.3 ± 5.9 (7)	17.6 ± 18.7 (5)	4.3 ± 0.9 (6)	2.2 ± 1.5 (9)	23
Heart	8.8 ± 5.4 (6)	2.7 ± 0.4 (7)	2.2 ± 0.7 (5)	1.6 ± 0.3 (6)	1.1 ± 0.3 (9)	<7
Tail muscle	5.1 ± 1.8 (6)	1.6 ± 0.1 (7)	1.3 ± 0.3 (5)	0.8 ± 0.2 (6)	0.4 ± 0.2 (9)	<7
Claw muscle	5.0 ± 3.1 (6)	1.5 ± 0.2 (7)	1.3 ± 0.1 (5)	0.8 ± 0.1 (5)	0.6 ± 0.2 (9)	<7
Stomach	4.5 ± 1.1 (6)	4.8 ± 2.1 (7)	4.3 ± 2.0 (5)	2.2 ± 1.0 (6)	1.3 ± 0.4 (9)	28
Gill	3.4 ± 1.8 (6)	3.2 ± 1.5 (7)	3.1 ± 2.3 (5)	1.4 ± 0.9 (6)	0.8 ± 0.3 (9)	24
Male gonad	8.3 (1)	1.6 (1)	2.9 (2)	2.1 (2)	1.1 ± 0.2 (5)	<7
Egg masses	16.6 ± 9.7 (5)	7.7 ± 6.6 (6)	10.2 ± 6.0 (3)	8.6 ± 4.0 (6)	7.7 ± 4.0 (4)	39
Plasma	1.33 ± 0.41 (6)	0.49 ± 0.17 (7)	0.62 ± 0.07	0.36 ± 0.13 (5)	0.20 ± 0.05 (9)	<7
Feces	--	--	142.5 ± 70.7 (5)	21.5 ± 6.6 (6)	20.1 ± 10.5 (9)	

[a] Data from Bend et al. (ref. 12).

[b] Mean ± SD (N).

Table 3. Percent administered radioactivity remaining in tissues of lobster with time after pericardial injection of ^{14}C-2, 4, 5, 2^1, 5^1-pentachlorobiphenyl (0.2 mg/kg)[a]

Tissue	Time after injection				
	24 hr	1 wk	2 wk	4 wk	8 wk
Hepatopancreas	61.4 ± 8.3 (6)[b]	60.5 ± 10.8 (7)	61.4 ± 14.6 (5)	55.2 ± 13.4 (6)	31.2 ± 12.5 (9)
Green gland	0.19 ± 0.12 (6)	0.07 ± 0.01 (7)	0.07 ± 0.03 (5)	0.05 ± 0.01 (6)	0.04 ± 0.02 (9)
Intestine	0.10 ± 0.09 (6)	0.11 ± 0.05 (7)	0.21 ± 0.27 (5)	0.04 ± 0.01 (6)	0.03 ± 0.02 (9)
Heart	0.09 ± 0.04 (6)	0.03 ± 0.01 (7)	0.04 ± 0.02 (5)	0.02 ± 0.01 (6)	0.01 ± 0.01 (9)
Tail muscle	4.36 ± 0.84 (6)	1.57 ± 0.26 (7)	1.54 ± 0.41 (5)	0.96 ± 0.26 (6)	0.45 ± 0.02 (9)
Stomach	0.36 ± 0.14 (6)	0.53 ± 0.39 (7)	0.45 ± 0.22 (5)	0.20 ± 0.10 (6)	0.12 ± 0.04 (9)
Gill	0.61 ± 0.31 (6)	0.59 ± 0.31 (7)	0.60 ± 0.41 (5)	0.25 ± 0.15 (6)	0.16 ± 0.07 (9)
Male gonad	0.06 (1)	0.02 (1)	0.03 (2)	0.04 (2)	0.01 ± 0.01 (5)
Egg masses	0.48 ± 0.34 (5)	0.37 ± 0.56 (6)	0.46 ± 0.44 (3)	0.64 ± 0.38 (4)	0.67 ± 0.53 (4)
Mean total recovery	67.6%	64.8%	64.8%	57.4%	32.7%

[a] Data from Bend et al. (ref. 12).

[b] Mean ± SD (N).

extractions with each solvent), but about 70 percent of the organic extractable material was chromatographically identical to 2,4,5,2′,5′-PCB. Neither the water soluble fecal metabolites nor green gland metabolites were investigated further.

Total recovery experiments in which lobster carcasses were homogenized after dissection accounted for as much as 90 percent of the injected radioactivity.

The specific radioactivity (dpm/mg tissue) was higher in dogfish shark liver than in any other tissue following parenteral administration of ^{14}C-2,4,5,2′,5′-PCB (table 4). There was an apparent increase in specific radioactivity of liver between 6 and 24 hours, concomitant with a statistically significant decrease in the radioactivity of most other tissues. Liver radioactivity remained at, or above, the 6-hour level throughout the course of the experiment (20.3 dpm/mp at 12 days, the longest period sharks could be maintained).

At all times subsequent to administration of ^{14}C-2,4,5,2′,5′-PCB to dogfish sharks, almost all of the recovered radioactivity was located in the liver (table 5), and initially all of the benzene extractable activity from hepatic tissue was unchanged ^{14}C-2,4,5,2′,5′-PCB. Only trace amounts of radioactivity were present in all other tissues (less than 1 percent) with the exception of muscle which contained 7 to 9 percent of the dosed activity 3 and 7 days after treatment. This observation was attributable more to the large muscle content of the dogfish (about 23 percent) (ref. 14) than to the specific radioactivity of the tissue.

Urine was collected continuously from a few sharks with cannulated urinary papillae. Only trace amounts of radioactivity were excreted in the urine of these fish after injection of ^{14}C-2,4,2′,5′-PCB. The delayed biliary excretion of radioactivity and the excretion of only very small quantities of biliary metabolites suggested that dogfish sharks biotransformed the dosed compound very slowly. Unaltered 2,4,5,2′,5′-PCB was not excreted in the bile and the biliary radioactivity was not converted to organic soluble material by β-glucuronidase hydrolysis indicating that glucuronide conjugates of phenols were not major biliary metabolites.

The decline in blood radioactivity following the intravenous administration of ^{14}C-2,4,5,2′,5′-PCB is illustrated in figure 1. The disappearance of radioactivity follows a multiple exponential decay relationship with time. Using least squares technics (fig. 2), we resolved this blood decay curve into three components by computer analysis. The $t_{1/2}$ of ^{14}C-2,4,5,2′,5′-PCB was estimated for each component; the α, β, and γ components of the radioactivity disappearance curve had $t_{1/2}$ values of 4, 27, and 730 minutes, respectively. It is interesting that the α and β components for blood disappearance of the PCB isomer tested had similar $t_{1/2}$ values to those reported by Dvorchik and Maren (ref. 14) for DDT blood disappearance in this same species (3 and 30 minutes, respectively).

The initial, rapid phase of radioactivity removal from blood after injection of ^{14}C-2,4,5,2′,5′-PCB is probably related to the rapid uptake into liver.

Table 4. Specific activity of various dogfish shark tissues and fluids with time following intravenous injection of ^{14}C-2, 4, 5, 2^{1}, 5^{1} -pentachlorobiphenyl (0.03 mg/kg)[a]

Tissue	Time after administration			
	6 hr[b]	24 hr[b]	3 days[b]	7 days[c]
	dpm/mg tissue or /µl fluid			
Liver	14.00 ± 2.15	21.63 ± 4.80	18.98 ± 5.77	14.05 ± 2.39
Kidney	5.39 ± 0.72	1.04 ± 0.22[e]	0.65 ± 0.05[e]	0.49 ± 0.05[e]
Salt gland	3.12 ± 0.91	0.72 ± 0.07[e]	0.60 ± 0.07[e]	0.36 ± 0.05[e]
Pancreas	2.31 ± 0.77	0.49 ± 0.08[e]	0.53 ± 0.16[e]	0.33 ± 0.05[e]
Spleen	1.86 ± 1.04	0.59 ± 0.05[e]	0.61 ± 0.08[e]	0.46 ± 0.06[e]
Gill	1.84 ± 0.38	0.78 ± 0.05[e]	0.55 ± 0.13[e]	0.70 ± 0.21[d]
Brain	0.92 ± 0.30	1.07 ± 0.24[e]	0.40 ± 0.16[e]	0.16 ± 0.03[e]
Heart	0.82 ± 0.10	0.36 ± 0.02[e]	0.41 ± 0.11[e]	0.15 ± 0.04[e]
Muscle	---	---	0.87 ± 0.42	0.66 ± 0.08
Blood	0.80 ± 0.02	0.32 ± 0.08[d]	0.50 ± 0.23	0.14 ± 0.13[e]
Bile	0.41 ± 0.21	1.35 ± 0.51[d]	2.71 ± 0.58[e]	12.03 ± 4.83[e]
CSF	0.02 ± 0.03	0.03 ± 0.05	0.06 ± 0.05	0.04 ± 0.04

[a] Data from Hart et al. (ref. 13).

[b] Mean ± SD (N = 3).

[c] Mean ± SD (N = 4).

[d] $p < 0.05$ compared with 6 hr.

[e] $p < 0.01$

Table 5. Percent administered radioactivity remaining in tissues of Dogfish shark with time after intravenous injection of ^{14}C-2, 4, 5, 2^{1}, 5^{1} -pentachlorobiphenyl (0.03 mg/kg)[a]

Tissue	Time after injection			
	6 hr[b]	24 hr[b]	3 days[b]	7 days[c]
Liver	75.22 ± 6.11	94.02 ± 24.65	79.55 ± 12.77	89.42 ± 11.17
Kidney	0.43 ± 0.16	0.06 ± 0.01	0.06 ± 0.01	0.06 ± 0.02
Salt gland	0.08 ± 0.02	0.02 ± 0.002	0.01 ± 0.001	0.01 ± 0.001
Pancreas	0.25 ± 0.03	0.05 ± 0.02	0.05 ± 0.02	0.03 ± 0.004
Spleen	0.21 ± 0.04	0.08 ± 0.02	0.06 ± 0.02	0.07 ± 0.03
Brain	0.03 ± 0.01	0.03 ± 0.01	0.01 ± 0.001	0.01 ± 0.002
Heart	0.05 ± 0.01	0.01 ± 0.002	0.02 ± 0.004	0.01 ± 0.003
Mean total recovery	76.3%	94.3%	79.8%	89.6%

[a] Data from Hart et al. (ref. 13).

[b] Mean ± SD (N = 3).

[c] Mean ± SD (N = 4).

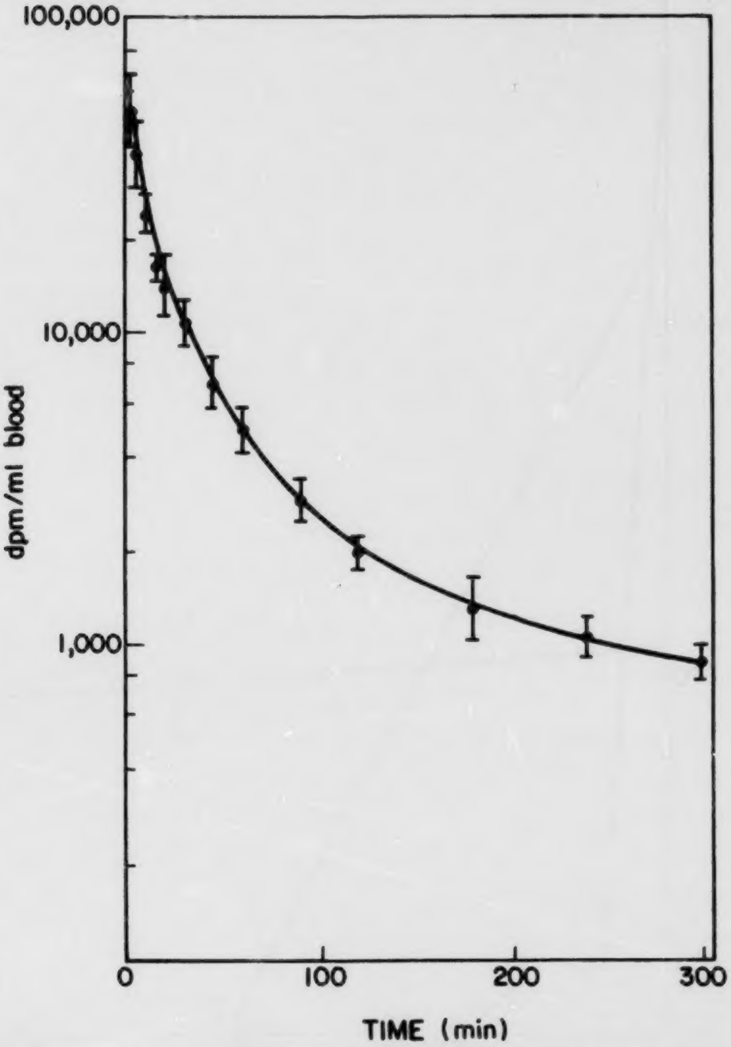

Figure 1. Decline in blood activity following the intravenous administration of ^{14}C-2, 4, 5, 2^1, 5^1-PCB.

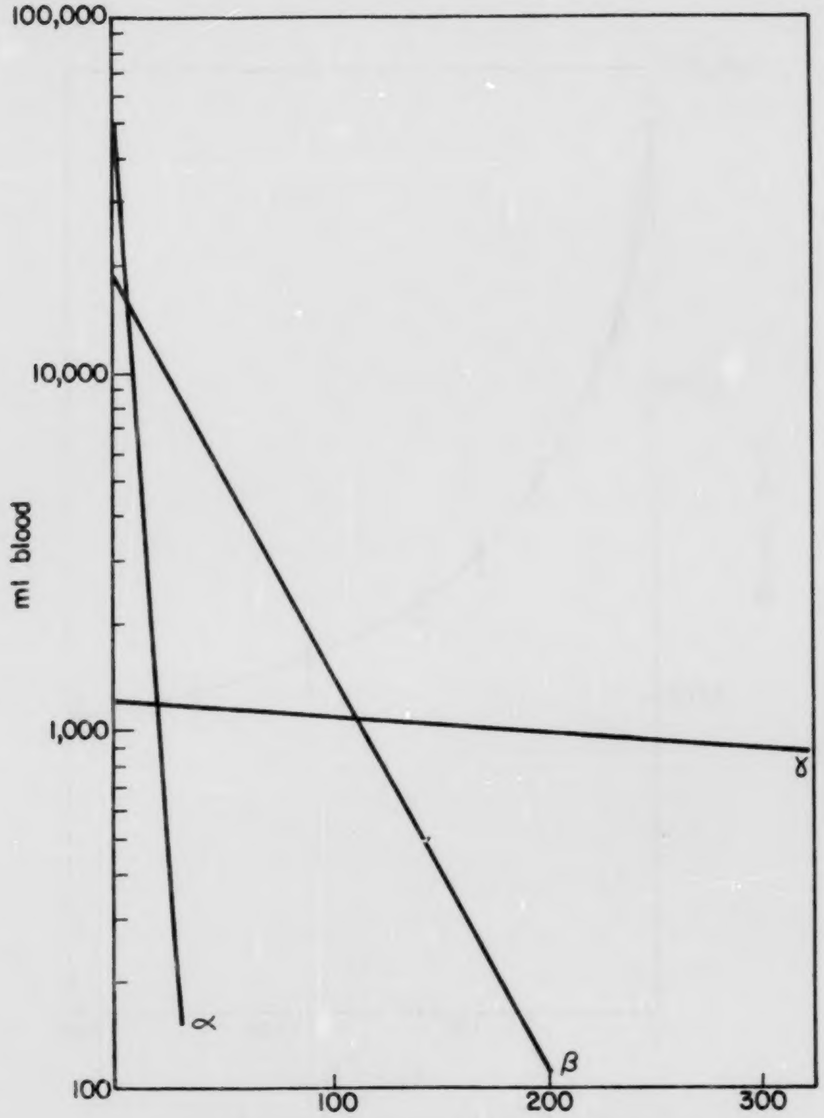

Figure 2. Three components of blood decay curve.

Microsomes prepared from lobster hepatopancreas had no detectable aniline hydroxylase, benzphetamine N-demethylase, 7-ethoxycoumarin 0-deethylase, or benzpyrene hydroxylase activity (table 6), whereas dogfish shark liver microsomes had appreciable (but low, relative to the little skate, *Raja erinacea*, another marine elasmobranch indigenous to Maine) mixed-function oxidase activity toward each substrate tested. These assays were run at 30° C, the optimal in vitro temperature for 7-ethoxycoumarin 0-deethylation in the dogfish (ref. 8). At 12° C, which is within the range of summer water temperature in Maine, specific 7-ethoxycoumarin 0-deethylase activity was only about 20 percent of that found at 30° C.

DISCUSSION

Although the accumulation and toxicity of commercial PCB mixtures, consisting of many isomers of varying chlorine content, were demonstrated in many aquatic species (refs. 15-22) few such studies have utilized single, purified PCB isomers. Hutzinger et al. (ref. 23) demonstrated that 4-chlorobiphenyl, 4,4′-dichlorobiphenyl, and 2,2′,5,5′-tetrachlorobiphenyl were hydroxylated by rat and pigeon but not by brook trout, a freshwater species. No hydroxylated metabolites of 2,2′,4,4′,5,5′-hexachlorobiphenyl were found in rat, pigeon, or brook trout.

In previous studies from our laboratory, we found that many marine species have some hepatic microsomal mixed-function oxidase activity, but that this activity is generally considerably lower than that found in mammalian liver (refs. 8, 24-26). Reduced rates of oxidative xenobiotic metabolism in marine vertebrates and invertebrates would partially account for the persistence of lipophilic compounds, such as the PCB's, in these species.

The distribution, excretion, and metabolism of ^{14}C-2,4,5,2′,5′-PCB in the rat was studied by Matthews et al. (refs. 27-31), working in our laboratory. This compound is metabolized readily by rats and the major metabolites are 3-hydroxy-, 3′,4′-dihydroxy-, and 3′,4′-dihydro-3′,4′-dihydroxy-2,4,5,2′,5′-pentachlorobiphenyls; the first half-life in rats is only about 2.5 days. This varies markedly from both the lobster (tables 2 and 3) and the dogfish shark (tables 4 and 5), where excretion of radioactivity occurs very slowly and the fatty hepatopancreas and liver, respectively, act as storage depots for ^{14}C-2,4,5,2′,5′-PCB.

The very slow metabolism (and excretion) of ^{14}C-2,4,5,2′,5′-PCB in the lobster is probably related to the very low microsomal mixed-function oxidase activities of lobster hepatopancreas (table 6). In separate experiments, we found only traces of benzpyrene hydroxylase or aniline hydroxylase activity in gill or hepatopancreas homogenate. The much lower concentrations of PCB's (tables 1 and 2) in lobster muscle and dogfish muscle (vs. hepatopancreas or egg mass) suggest that portions of marine species might be used for foodstuffs even in certain organs exceed the FDA recommended

Table 6. Mixed-function oxidase activity in lobster hepatopancreas and dogfish shark liver microsomes[a]

Species	Aniline Hydroxylase[b]	Benzphetamine N-demethylase[b]	7-Ethoxycoumarin 0-deethylase[b]	Benzpyrene Hydroxylase
Lobster, *Homarus americanus*	0.01 (3)[d,e]	0.06 (3)	0.01 (3)	0.01 (3)
Dogfish Shark, *Squalus acanthias*	0.07 ± 0.01 (3)[f]	0.15 ± 0.05 (3)	0.08 ± 0.02 (3)	0.07 ± 0.02 (3)

[a] Data taken from Pohl et al. (ref. 8).

[b] Nmoles/min/mg microsomal protein.

[c] Fluorescence units/min/mg microsomal protein.

[d] Minimum metabolism detected by assay procedure used.

[e] Microsomes from three lobsters were tested separately.

[f] Mean ± SD (N).

PCB levels, provided that care is taken in preparation and that no PCB metabolites or degradation products with high toxicity to humans are present (ref. 32).

Although hepatic microsomes from dogfish shark have mixed-function oxidase activity, this activity is low relative to mammals and some other marine species (ref. 8). Furthermore, dogfish liver is very fatty and the microsomal protein yield is very low (only 1-3 mg protein/g liver vs. 6-15 mg/g liver in the little skate), meaning that whole liver xenobiotic-metabolizing activity per gram tissue in the dogfish is even lower relative to the skate than is specific activity (per mg microsomal protein).

The liver appears to function both as the major site of ^{14}C-2,4,5,2′,5′-PCB uptake and storage in the dogfish shark, probably due to its high lipid content. The delayed excretion may be related to poor partitioning of the compound from liver lipid into metabolically active parenchymal cells, and to the low mixed-function oxidase activity, especially at 12° to 15° C which is the Maine water temperature in summer.

The persistence of trace doses of ^{14}C-2,4,5,2′,5′-PCB, an isomer that is metabolized rapidly by rats, in the two marine species studied suggests that the problem of PCB residues in marine and freshwater species will be with us for some time, especially in the face of continued PCB release into the aquatic environment.

ACKNOWLEDGMENT

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20 November 1975

Session V:

ECONOMICS AND SUBSTITUTES

Warren Muir, Ph.D.[*]
Session Chairman

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INTRODUCTORY REMARKS

Warren Muir, Ph.D.

I guess we have all had an opportunity to hear about health and ecological impact and other aspects of PCB's in the last day and a half. The history of PCB's, as I am sure most of you are aware, is several years old. By 1971 and 1972 there was sufficient concern over their potential environmental effects that a Federal task force was created under the sponsorship of the Council on Environmental Quality and the Federal Council for Science and Technology, which looked into the situation and made a variety of recommendations.

In addition, at that time the Monsanto Chemical Company developed a restricted sales policy with regard to uses of PCB's, and also shortly thereafter, the Organization for Economic Cooperation and Development had an international agreement with regard to uses of PCB.

All of these actions were directed in one fashion or another toward inappropriate uses of the chemical. Yet, as of this date, there is no Federal authority to control the use of these chemicals, despite 5 years of consideration of toxic substances control legislation.

This particular session of the conference deals with economics and substitutes; we will have a presentation or a brief description of two economic case studies that have been done on PCB's, and then several speakers will discuss the substitutability or nonsubstitutability for uses of PCB's.

In dealing with environmental problems such as PCB's and a variety of other chemicals, I am sure you are aware that there is a great deal of scientific uncertainty with regard to particular health or ecological impacts. For example, there is a certain amount of uncertainty as to whether or not something is carcinogenic or whether it can bioaccumulate.

There typically appears to be, however, a much greater uncertainty about the economic implications or the economic impacts of any action that might be taken to mitigate the suspected problems. Much of this arises from uncertainty, about substitutes and also about the inability to predict the actual individual or corporate responses that would be made under a given type of regulatory action or other type of mitigating action. Thus in my view, there is much greater economic uncertainty in many of these issues than there is scientific uncertainty.

Also, irrespective of all the uncertainties, we know that the further along in the chemical marketing and development process, the longer it has been manufactured, and the larger the market, generally the greater the health and environmental effects of the particular chemicals and the greater the economic impact there is of mitigating action, to the extent that they are necessary.

Thus, if we are to deal with problems such as PCB's, we must change our approach toward environmental chemical problems; we must get out of a reactive posture. We must undertake the task of identifying potential hazardous chemicals early in the development process. By so doing, health and ecological problems can be avoided before they develop. And, by so doing, the economic dislocation and impacts from any potential regulatory action will be eliminated or minimized to the benefit of all.

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PCB's IN CAPACITOR APPLICATIONS

Richard L. Rollins*

Abstract

Alternating current (AC) capacitors have used a grade of polychlorinated biphenyls (PCB's) since the 1930's. Long life, high reliability and a high degree of flame retardancy are imparted to the capacitors by these fluids.

The latter characteristic is very important in applications where high exposure to the general population exists such as in fluorescent lights and television sets. The main disadvantage with the fluid is its long persistence in nature. When the capacitor industry became aware of the environmental problem, it generated guidelines for handling and disposal of the material, then voluntarily imposed these restrictions upon itself. Now of the 70,000 pounds per day processed, only 7 pounds are being discharged into plant water effluents. Liquid waste materials are sent to proper incineration facilities and solid waste materials are placed in sanitary landfills to prevent escape into the environment. For 4 years the industry has been using a significantly more biodegradable grade of Aroclor identified as 1016 by Monsanto. In that time, Aroclor 1016 has not been identified as a PCB commonly found in the environment. Non-PCB materials are continuing to be evaluated, but there are not dielectric fluids available today which can be considered an acceptable substitute for PCB's in the broad range of AC capacitors.

On behalf of the electronic industrial association and the manufacturers of PCB capacitors, I appreciate this opportunity to present our point of view. I will discuss PCB capacitors, the industry's response to the environmental problem, and possible alternative candidates to PCB's.

Manufacturers of AC capacitors have used PCB's since the early 1930's, and have used them almost exclusively since World War II. They are presently used, for example, in capacitors for fluorescent lighting, air conditioners, and television sets. These capacitors are in the plant where you work, in the home, and in public gathering facilities. They are therefore near you a high percentage of the time. Manufacturers are very cognizant of their responsiblities in providing a capacitor that will operate satisfactorily in these applications for providing long life and high reliability at a reasonable cost.

The consequence of these requirements in a competitive marketplace is that the products and their components are constantly being reevaluated to increase life, increase reliability, decrease size, and decrease cost. The capacitor manufacturer indeed daily sees pressures for improvements. It is, therefore, an obvious question to ask why are PCB's still used after 40 years during which the best capacitor research and development scientists in the United States were constantly seeking substitutes.

One of the reasons lies in the nonflammability of the PCB material. Because of applications, such as lighting, where personnel and equipment safety are paramount, capacitors must be manufactured since they not only fail infrequently but also fail safely. In applications such as the World Trade Center in New York, where 250,000 fluorescent lights are installed, it is obvious that high reliability and a nonflammable capacitor characteristic are mandatory.

A capacitor which fails violently and contains a flammable fluid could create a serious problem, especially in densely populated buildings, giving visions of a towering inferno. The Consumer Products Safety Commission, as an example, has shown considerable concern over television set fires and has requested Underwriters' Laboratories to develop standards for safety for television set receivers.

A second reason lies in the reliability of the product as it is known today. Presently, the capacitors our companies manufacture have a survival rate per year greater than 99.9988 percent. The requirement that more than 95 percent of our capacitors must survive after 13 years of normal application conditions must also be met.

The test time and the amount of test necessary to guarantee the above reliability is mind boggling to say the least. Many thousands of units and millions of unit hours at or referred to application conditions are required to satisfy our statisticians that any change in product will conform to these present day standards.

Even with the large amount of previous testing and history on the PCB-containing capacitor, there have been many instances which despite all controls in existence cause consumer concern due to extreme failure rates. That is at a rate greater than 10 percent.

The first occurred in 1957, when a variation in the quality of the PCB fluid, as synthesized, resulted in a highly unstable capacitor. Standard analytical tests in use at the time did not detect the quality difference. The result was a large number of capacitors failing in a very short period of application life.

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The consequence was the requirement by the customer that the capacitor manufacturer not only provide compensation for the cost of the capacitor, but the cost of the equipment which contained the capacitor, plus the labor charge for placing the equipment in the installation. The total replacement cost the manufacturer 100 times the original price of the capacitor.

The second instance of numerous failures occurred 6 years later, this time involving the capacitors of most manufacturers. In this circumstance reduced-size capacitors were tested and approved by industry-accepted accelerated life testing and a new size was shipped to customers.

Approximately 1 year later, up to 15 percent of these capacitors began failing in some applications. A subsequent modification in the accelerated-life testing based upon findings in the field failure analyses now provides proper screening to eliminate the chance of such a problem occurring. Therefore the AC capacitor industry now does very extensive evaluations before releasing new designs or using new materials.

The capacitor industry became aware that PCB's were possible environmental problems in 1970. Soon thereafter, in 1971, a committee was formed under the sponsorship of the American National Standards Institute, ANSI, to develop standard industry guidelines for the handling and disposal of capacitor grade PCB's. An interim standard was published as an official standard proposal by the National Electric Manufacturers Association, NEMA, in January 1973. And a final document, C107.1 1974, was published in January 1974.

Briefly, properly planned housekeeping, considerations for employee safety, and scrap disposal procedures were detailed. Today, to the best of my knowledge, all manufacturers of PCB capacitors in the United States have applied these guidelines for the handling and disposal of PCB's.

The success of the manufacturer's control in handling practices is apparent from the fact that of the approximately 70,000 pounds per day used, less than .01 percent or 7 pounds per day is discharged into the water effluents of the plants. The ANSI guidelines also recommended that the capacitor companies change from Aroclor 1242 to a PCB with less toxic effects which Monsanto identified as Aroclor 1016.

Here it must be pointed out that various grades of PCB's cannot, as so often is assumed, be classified as having the same chemical, physical, or biological characteristics. For instance, in "A Comparative Study of Two Chlorinated Biphenyl Mixtures . . .," by Goldstein et al., significant differences were shown to exist in the biological effects of Aroclor 1242 and 1016 on equal doses of the two mixtures. Aroclor 1016 also reduces by greater than 94 percent those higher homologs existing in Aroclor 1242 which were the most persistent in nature. With these benefits, and without sacrificing the capacitor characteristics, all capacitor manufacturers began using the new material by 1972, despite the fact that it was 50 percent more expensive.

After 4 years of use by the capacitor industries, Aroclor 1016 has not been identified as a PCB that is commonly found in the environment. Nor should it be suspected that 1016 would be commonly found, because the industry is exercising control over waste disposal from its capacitors, so that there is little or no opportunity for leaking PCB's.

Our industry believes it is much more important to reduce the environmental problems and to allow continued use of the capacitor-grade PCB than to be concerned with the material cost increase and immediately turn to a substitute which is not thoroughly evaluated and may be flammable.

Alternates have been considered ever since the original introduction of PCB's. The substitutes must be not only a satisfactory dielectric fluid providing acceptable life, reliability, and safety, but in addition must not cause environmental problems. Our experience with PCB's has demonstrated that some material will escape into the environment through processing and handling. Therefore, alternate fluids must be evaluated on this basis. The effects on man and his environment must be determined before the product is introduced. It would be a very serious error to replace capacitor-grade PCB's with a fluid which eventually becomes a greater threat to man.

Further PCB modification which would provide even less toxicity and persistence in nature but still maintain good dielectric properties and good nonflammability would seem to be one alternative to Aroclor 1016. Our companies respectfully urge Monsanto to pursue this possibility.

Non-PCB alternatives are now being offered and suggested. Some are new and some have been available for many years. The industry's position on these is as follows.

Mineral oil is a flammable fluid which was replaced by PCB's in the 1930's. Capacitors using it are 50 to 100 percent larger, much less reliable, and much less safe than those using PCB's. The substitution of mineral oil necessitates redesign of equipment to utilize the larger capacitor, an increase in the capacitor manufacture facility, and will result in increased use of basic materials which are presently in short supply.

Modified synthetic hydrocarbon oils have been developed which while flammable would allow capacitors to approximate today's size. Samples have been distributed for testing, but the material is not commercially available.

Phthalate ester, also a flammable fluid and also with biodegradability problems, has been used in certain restricted applications where conditions of limited temperatures exist. The unknown factors of reliability and safety in the broad consumer use areas such as lighting now prevent its use. Evaluations, however, are continuing.

Substituted aromatic compounds are possible candidates, and one has been proposed that has less fire resistance than PCB's; it is suggested to be a good dielectric fluid but only for high-voltage power factor correction. This, to our estimation, represents only 17 percent of the PCB used in closed systems, and leaves completely unanswered an alternative or alternatives for the remaining 83 percent of the applications. Some material has been produced but the fluid has not been made commercially available. In fact, our companies are having trouble obtaining samples from the manufacturer. When samples become available, the time-consuming testing can begin. It has been estimated that 3 years are required before final commercial use after initial testing of a change in capacitor fluids.

Also proposed for limited applications is the use of a plastic film replacing the kraft paper dielectric in the AC capacitor. Although evaluations with the highest quality film have been underway for more than 2 years using the current processing techniques, the reliability of the product is only 1/20th of that of present capacitors in most applications. Significant additional testing is required to determine if the cause of poor reliability is totally inherent or if improvements can be made by modifications in capacitor design.

In summary, there are no commercially available fluids which today can be considered a totally acceptable substitute for PCB's in the broad range of AC capacitors, nor are there substitute dielectric systems which would satisfy the requirements of reliability and safety in most applications.

I would like to reiterate that our companies have shown responsiveness to the environmental problems of PCB's by the following actions.

1. Working as a group with government and consumers in providing guidelines for proper handling and disposal of capacitor- and transformer-grade PCB's.
2. Reducing discharge levels of PCB's in a 16-capacitor plant. Further reductions are now being planned.
3. Converting to a more expensive grade of PCB's which is less persistent and has less toxic effects.

Finally significant differences do exist between Aroclor 1016, which capacitor manufacturers are now using, and material which is predominantly being found in the environment. Our industry believes that all concerned should be aware of the differences.

We respectfully urge this awareness in environmental studies and in drafting of regulations.

THE ECONOMIC IMPACT OF A BAN ON POLYCHLORINATED BIPHENYLS

Duncan MacArthur* and Stephen F. Nagy†

Abstract

The study described below assessed the effects of a hypothetical total ban on PCB's. In order to compile a realistic scenario, the proposed Toxic Substances Control Act was used as a model. A complete phaseout was estimated to take 76 months, and would involve publication of rules, testing, hearings, halting of imports and manufacturing of PCB's, and depletion of stocks of PCB-containing products.

Mineral oil was assumed to be the primary substitute for PCB's, since a major technological breakthrough in the development of alternate fluids is unlikely in the near future.

Minimum estimates are given for the one-time and annual capital expenditures by industry of a ban on PCB's, and for the primary and secondary impact costs. Intangible issues, such as the effect of a PCB ban on public safety, are also discussed.

In the course of our work with private and public clients, we have examined polychlorinated biphenyls, PCB's, in a number of areas, ranging from product design to economic impact. The work to be discussed here was completed in 1975 as part of a study to examine the effects of the proposed Toxic Substances Controls Act, as illustrated by Senate Bill S776 (20 Feb. 75).

We selected as an example a case study to completely ban an existing product following the procedures outlined in the proposed bill. We use the case study for following reasons. It provides an example of the complete analysis required to assess the impact of a ban; it illustrates the direct and secondary impact on industry; and it enables us to estimate the economic consequences of a ban on one specific product. In our study, we also highlighted the unquantifiable factors.

In our methodology, and again this was done in the early part of the year, we assume all the steps in the proposed Toxic Substance Control Act as illustrated by Senate Bill S776 are taken. The end result of this is a total ban on PCB manufacture and use. We selected PCB's for illustrative purposes only. Our discussion was not intended to reflect on the actual health or environmental aspects of the example product.

The scenario for banning the product is estimated to require about 76 months for complete phaseout of product use. Thirty-eight months were estimated for publications of the rules, testing, hearings, and so forth. The key element in this period is the 24-month period that we estimated would be required for testing. The key element in months 39 through 76 is phasing out the sale of some manufactured goods, particularly appliances. We also assumed that no legal action would be taken by industry until publication of the final ban on substances occurring after the 38 months of testing and hearings.

A possibility in the scenario of the second 38 months is that industry requests a judicial review but complies with the review. We estimated the judicial review takes 2 years and results in a complete product ban. In the second 38-month period the following types of activity would go on: documentation for control purposes; monitoring of shipments; limitation of stockpiling of PCB products; refining methods to prevent PCB's from entering the environment, such as controlled use and control proposal; stopping the production and import of PCB's or equipment containing PCB's, and finally banning the manufacture and sale of PCB-containing products.

In the analysis of our PCB's we have tried to consider the complete life cycle of the product. PCB's are produced by one manufacturer in the United States—the trade name is Aroclor—and about 40.5 million pounds were produced in 1974. The sale of PCB's is currently restricted to electrical use and casting waxes. In the past they were used as transfer fluids from heat exchangers, hydraulic fluids, and so forth. However, as you know, to avoid potential adverse environmental impact, Monsanto restricted domestic and export sales in 1972 to electric installation equipment applications in which the product is enclosed in a relatively well-maintained container throughout its use. Monsanto and others offer PCB disposal service.

We found that PCB applications are characterized by their long-term utilization with the majority controlled by utilities. Transformers, for example, were reported to last 20 to 30 years, and capacitors to last 7 to 10 years.

The number of transformer manufacturers vary; anywhere from 2 to 10 have produced PCB-containing transformers. Primary use is as nonflammable electric insulating oil. Previous estimates report approximately 5,000 transformers were produced per year with an average PCB amount of about 5,000 pounds per transformer. Value of shipments (1974 data) is estimated at $35 to $45 million.

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The number of people using capacitors varies. Estimates range from 16 up to 50 capacitor manufacturers. There are approximately 90 to 100 million units per year produced with a value about $105 to $147 million. There are primarily two types: large utility capacitors, using about 9 to 13 million pounds of PCB's per year, and small industrial capacitors, using about 17 million pounds per year.

In the case of casting wax, there is only one manufacturer in the United States. PCB's represent 30 percent of the components of the wax. The annual usage is about half a million pounds. Primarily, it is imported. We also found another indirect importation of PCB's—in capacitors in electrical equipment.

The primary transformer users are utilities; others include manufacturers of and users of electrically powered rail equipment, furnace equipment, and electrostatic precipitators. About 85 percent of PCB transformers are used as network transformers by the utilities, and we estimate that approximately 90 percent of the transformer uses are controlled by the utilities, either through ownership or service contracts.

With respect to the capacitor users, electric utilities use large capacitors and manufacturers and consumers of goods use small capacitors. Electric utilities use them primarily for power factor correction, and smaller units are used for starting and other uses such as fluorescent lighting. We estimate that about 2 to 3 percent of large capacitors are consumed as replacements and about 5 to 10 percent of the small capacitors were consumed as replacements.

With respect to the casting wax, approximately 98 percent of that is now recycled and the people in the business maintain that the remaining 2 percent that is left in the mold is destroyed when the mold is prepared at high temperatures.

The final step is disposal. Monsanto and others have specialized incinerator facilities for PCB liquids, primarily for large uses such as transformers and capacitors used by utilities.

The ban of PCB's was estimated to result in a one-time cost of $13.7 million. There would be additional expenditures of $110 million annually. The ban would also affect regulatory codes, public safety, and other intangible areas, and, as always, the net result would be an increase in consumer costs.

The primary impact, which is on the manufacturer and users, is estimated at $8.8 billion for one-time costs and approximately $16 million for annual costs. Key elements in this, surprisingly enough, were "red tape" required by Senate Bill S776. There are also disposal costs, plant rebuild costs, and salary costs.

Secondary impact on users of PCB's, PCB-containing products or services, and disposal organizations is estimated at $4.9 billion for one-time costs and $93 billion for annual costs. Elements of secondary impacts are primarily new plant rebuilds for increasing the capacitor output of the United States, disposal, and the increased cost of substitutes.

The annual costs reflect the forecasted increased costs of electrical equipment manufacture and use, including replacements of existing equipment. Since the lifetime of large transformers and capacitors is 20 to 30 years, the additional annual cost, over $110 million, was forecast for the same period.

In the proposed 38-month time period, industry representatives indicated that for primary and secondary transformer use, there would essentially be a shift to mineral oil as a replacement for PCB's, since the environment and performance characteristics of other substitutes are not thoroughly researched. Major technological breakthroughs, such as development of substitute fluids, were not forecast.

We did use minimum cost estimates. The cost of replacing a PCB transformer, for example—not necessarily the transformer but the vault which is surrounded by the building—has been estimated as varying anywhere from $5,000 to $50,000. We used a figure of about $10,000, which we consider low and very conservative.

The economic impact of changing the regulatory codes cannot be accurately quantified without a detailed survey. However, the impact of the changeover for those localities with regulations was estimated not to cause major dislocation in building activities. We do know that many cities permit only nonflammable, essentially PCB, electrical equipment to be installed in tall buildings; however, others do permit non-PCB transformers with a vault.

Intangible issues include public safety, which would be impacted by a ban on PCB's. Analysis would require a detailed analysis of the tradeoffs between the benefits of the product versus the benefits of the ban. Electrically driven trains use PCB transformers and it has been reported that the flammable liquid in the transformer would be a potential hazard. Uses of other transformers in rail cars, if possible, would require redesign and overhaul of existing equipment. Use of large quantities—over 20 gallons of liquid—in buildings would present a potential danger even in walls. Insurance underwriters have reportedly studied the problem but nothing has been presented as a request to change rates.

Other intangible areas are in replacements for all types of equipment. Dry transformers, for example, are reported to have a low reliability. There are also specific areas, such as replacement capacitors for compact equipment in which the capacitors cannot be replaced with a non-PCB-containing unit similar in size and characteristics, forcing either salvage or scrappage of the equipment.

In assessing all these costs, we forecasted pass-through to the consumer.

THE USE OF DOW CORNING® Q2-1090 DIELECTRIC LIQUID IN POWER TRANSFORMERS

Richard H. Montgomery*

Abstract

Dow Corning has developed Q2-1090 Dielectric Liquid to replace askarels in transformers. Five years of laboratory and field testing have shown it to be both efficient and safe. It is now being sold in commercial quantities both for new transformers and for retrofilling old transformers. With some redesign, competitive entries, and further restrictions on PCB use anticipated, it is expected to have favorable economics. A 3- to 5- year phase-in to completely commercially qualify the liquid in all applications is planned.

Thank you for the opportunity to come here today to represent the silicone industry, which in the United States consists of the Dow Corning Corporation, the General Electric Corporation, Union Carbide, and Stauffer Chemical. The global silicone industry is now 5 years into a program to qualify dielectric fluid replacements in both transformers and capacitors. The Japanese silicone industry and Dow Corning Corporation have led this effort to date. As a result of this extremely large effort by the industry, I am very pleased today to make two major announcements.

First, the technology to commercially manufacture a capacitor dielectric fluid with superior dielectric properties is being developed. This material appears to withstand any of the high electrical stresses the material would normally be used for in the industry. We plan to make this product commercially available during 1976. At its present state of development, we know of no environmental problems with this particular product. No chemistry would indicate there should be any. But this will be continually studied, as have other products from the silicone industry.

Second, Dow Corning now has commercial production available to manufacture silicone transformer liquids in sufficient capacity to handle the global transformer market, and that is a very large investment. So let me now turn in some detail to the transformer industry and the role silicones can play.

We must remember that today three environmentally safe transformer systems are available to replace transformers filled with PCB liquids. Gas-cooled, air-cooled dry types, and liquid-silicone-filled transformers will meet almost every need where PCB-containing transformer fluids are used.

The silicone industry has produced insulating materials for transformers for 25 years. The industry's newest material, a silicone transformer liquid, is now in use in Japan as a direct replacement for PCB-containing transformer oils. In the United States, extensive field testing has been under way for 4 years and at the present time this material is being examined for inclusion in the U.S. National Electrical Code.

Now, to underscore Dow Corning's personal assurance in the safety, the efficacy, and the efficiency of this liquid, Dow Corning has notified its vendors that we will be specifying silicone-filled transformers for all the new transformers in our production plants around the world. Our existing askarel transformers, in which we have a very heavy investment, are being phased out rapidly through a silicone retrofit program. Over 6 transformers have been installed new or retrofitted in the past year, and this program will accelerate in the spring. It is, of course, not possible in Michigan during the winter months to drain the PCB's satisfactorily and replace these materials with a silicone fluid.

The material that we are talking about is known today as Q2-1090 dielectric liquid. Basically, Q2-1090 transformer liquid is a dimethyl silicone that has been specially formulated and qualified for use in electrical applications. The technical feasibility of using the silicone in transformers has been shown in over 20 years of experience in military transformers, in several years of use on the Japanese National Railway, and in a number of power transformers in the Midland, Michigan, area; several other areas around the United States will also test silicone transformers in the next few months.

In addition to these actual applications, which today are well known and have worked out nicely, support for the use of a silicone dielectric fluid in power transformers is contained in over 30 years of accumulated data on dielectric properties and compatibility with the common materials of transformer construction.

The present concern for fire, explosion, health, and environmental hazards are all strong reasons for an evolutionary approach to using silicones and qualifying them in this application for all major power transformer uses over the next few years.

The silicone liquids of this type are much less flammable than typical mineral oils. They actually have a higher flash point than many PCB-containing transformer fluids. Although they will burn, they have an extremely low heat of combustion, extremely high flashpoints and firepoints, and they have been shown in catastrophic testing to be self-extinguishing. They appear to offer a level of fire safety greatly superior to mineral oils.

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The silicone that we are now recommending, a dimethyl silicone, has been evaluated by Underwriters Laboratory, and has received an extremely low flammability classification number. In Japan and the United States, transformer cases containing PCB's, silicones, and mineral oil were subjected to catastrophic failure testing. In both tests, all liquids exploded. Most important, following this test, only the mineral oil continued to burn. Both the polychlorinated biphenyl materials and the silicone fluid self-extinguished. Normally, in a test of this type you would fuse the circuit cutout. The fault in the silicone fluid self-cleared, thereby extinguishing the arc. With the other materials, the backup fuse blew and extinguished the arc.

Following the review of some very extensive data (which is available to all of you by writing to me at Dow Corning, provided you are willing to put in 4 or 5 hours in reading it), the major insurance companies have given their permission to use silicone fluid in indoor transformers. Because it is a new material and since no general policy has been established, the insurers have indicated that they will evaluate each application on an individual basis; this is a conservative approach which we highly applaud.

Dow Corning feels that we should look at a 3- to 5-year evolutionary approach of gradually putting a larger and larger number of transformers into operation. We are offering financial inducements to companies who do this in order to collect a large body of case history data that can be used to convince ourselves and the industry that there are no fatal flaws that have been overlooked. It's an extremely conservative approach.

Silicones are often used in small concentrations in the preparation of certain foods. Any time you eat a jam or jelly or drink a glass of some of that good old Milwaukee beer, you are eating a food-grade dimethyl silicone. And if you have an ulcer and you eat Di-Gel, you are also eating it. They are excellent deflaccuants.

The toxicity of dimethyl silicones to mammals, aquatic life, and plants has been very thoroughly investigated. The extremely low toxicity of silicones has made it difficult to detect any toxic reactions in test subjects. Studies directed toward determining the tendency of silicone to bioconcentrate have been negative. It has not been possible to date to show that any bioaccumulation was occurring.

It has been often stated that silicones are persistent in the environment because they do not biodegrade, under the evidence available at the present time. However, there is considerable evidence that silicones do chemically degrade in the environment. Contact with soil and water causes the liquid to depolymerize to low-molecular-weight species, and known chemistry definitely suggests that these degrade in water or in the atmosphere.

Silicones cost more money than the current liquids being used in transformers, but they are not outrageously expensive. Referring to the entire market of capacitors and transformers, and making some assumptions which would include the shipment of present products such as PCB's to the manufacturer's site, if we include the shipping costs, my estimate is that the industry PCB purchases would be about $19.2 million.

If good engineering practices are followed to reduce the amount of fluid needed, which has never been a design criteria in the major manufacturer's mind, as mineral oil, of course, is cheap, those costs would probably rise with the use of silicone to an industry figure of $25 million.

So the total increased cost to the electrical and electronics market would be about $5.8 million or 29 percent. If we made the assumption that the dielectric fluids cost is 20 percent of the total material cost going into a transformer or capacitor, we are talking about an average cost increase of about 5.8 to 6 percent at the manufacturing level on new equipment.

I feel that this slightly higher initial cost of silicone liquid-filled transformers is more than offset by the cost of monitoring and controlling of PCB-filled transformers over their lifetimes. And with silicone, no known environmental hazards are incurred.

PCB-filled transformers currently in service can be changed over to Q2-1090 transformer liquid without undue problems. I think the work we have done over the past 4 years—some of it in conjunction with Dow Chemical, a lot of it in conjunction with leading transformer manufacturers—shows this.

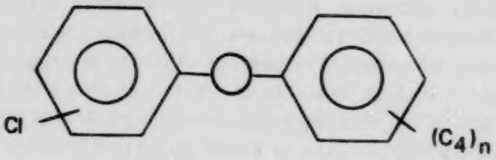
I think you will be hearing not only from Dow Corning in more detail on this subject. I highly suspect that our worthy competitors, who can manufacture dimethyl silicones to the same specifications once they learn what they are, will go into the market. At that particular time, the law of supply and demand will come into effect. The silicone industry at the present time has more capacity than it can sell. I anticipate the economics will be extremely favorable for further consideration of this product in both transformers and capacitors over the next three years.

DOW XFS-4169L: AN ENVIRONMENTALLY ACCEPTABLE CAPACITOR FLUID

Dean Branson, Ph.D.*

Abstract

Dow and McGraw-Edison Companies have developed a new capacitor fluid which is electrically, ecologically, and economically acceptable. Chemically, the new fluid can be described as butylated monochlorodiphenyl oxide.



n = 0, 1, 2, 3

Dow XFS-4169L capacitor fluid

Dow XFS-4169L capacitor fluid performs equal to or better than Aroclor 1016 in power capacitors. This conclusion is based on dielectric losses, discharge inception voltages, capacitor size, fire hazard, reliability, economics, and availability. Dow XFS-4169L capacitor fluid and its components are acceptable in terms of health and environment according to an assessment of biodegradability, bioconcentration in fish, toxicity to animals, and toxicity to fish. Other capacitor manufacturers are currently evaluating XFS-4169L for applicability in their respective companies. Field trails have been started with utility companies across the country.

The contents of this presentation represent 4 years of joint research between Dow and McGraw-Edison Company which has culminated in the development of an environmentally and electrically acceptable capacitor fluid.

When PCB's replaced mineral oils some 40 years ago in power capacitors, this represented another step forward in the electrical industry's continuing effort to provide the public with low-cost electrical power and safer electrical equipment. As a result of the use of PCB's, there has been a downward trend in the cost per kilovar of power factor correction.

In 1971, Monsanto limited the sale of PCB's for dielectric uses where acceptable alternatives were not yet available. Since then, industry has been searching for alternatives which were ecologically, electrically, and economically acceptable.

Today, we want to report to you that Dow and McGraw-Edison have developed a butylated monochlorodiphenyl oxide known as XFS 4169L. It meets all the above criteria and can be used in high-voltage capacitors.

Initially, over 50 fluids representing several chemical families were selected as possible dielectric fluids on the basis of their chemical and physical properties. These fluids were then screened for both electrical performance in miniature capacitors and for potential hazard to the health and the environment in a battery of indicative tests.

The conclusion of these screenings was that the most promising chemical family was the alkylated monochlorodiphenyl oxides.

Electrically, several members of this family performed equal to or better than Aroclor 1016. It was the health and environmental data that demonstrated the significant advantages of the butylated monochlorodiphenyl oxides.

Electrical Performance

The confidence that McGraw-Edison Company has in high-voltage capacitors made with the XFS fluid is based on the following assessment of these six key electrical performances.

1. *Dielectrical losses*. The dielectric losses are as low or slightly lower than for capacitors with a PCB known as Aroclor 1016. This is true for both paper-film and all-film high-voltage power capacitors. This means that the capacitors will operate under normal temperatures and that the operating costs will be minimal. This is one of the same significant advantages that PCB's have relative to mineral oil.
2. *Discharge inception voltages*. Discharge inception voltage is significantly higher than for Aroclor 1016 in both paper-film and all-film capacitors. This means that operating voltages may surge at least 20 to 30 percent higher without resulting in temporary malfunction due to electrical discharges known as corona.
3. *System size*. The size or volume per unit of high-voltage power capacitor correction known as kilovar is

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*Capacitor Fluid H.N.E., Project Manager, Health and Environment Research Laboratories, Dow Chemical, Inc., Midland, Michigan.

the same as for comparative capacitors impregnated with Aroclor 1016. This means that it will not be necessary to redesign capacitors, the capacitor manufacturing, or the application of capacitors.

4. *Fire hazards*. As indicated with PCB's, there is a low risk of explosion and fires from power capacitors impregnated with XFS 4169L. A major problem associated with a failure in a paper-film power capacitor is the decomposition of the paper and, to a lesser degree, the film. The resulting pressure of the gases formed is a major cause of tank rupture. After failure, the flammability of the fluid may contribute to a fire only if the paper, the film, and the fluid, are above the ignition or flash temperatures.

Under normal operating conditions, a capacitor will operate between 40° and 80° C. At a time of failure, this temperature will have to exceed the flashpoint of the capacitor fluid before this fluid becomes contributive to the explosiveness or the flammability of the system. In this regard, the flash temperature for the existing dielectric fluids for mineral oil is 154° C; for Aroclor 1016, 166° C; and for XFS 4169L, 174° C. The fire temperature for mineral oil, 167 PCB, is greater than 316° C; for XFL 4169L, it is 199° C. This clearly shows that the flash and fire points are substantially above the operating temperatures.

The National Electric Code allows the installation of the electrical devices without a vault if they contain less than 3 gallons of burnable liquid. The most common size of our capacitor used today is a 200-kilovar unit. These units contain less than 3 gallons of fluid, and are installed predominantly outdoors.

5. *Capacitor reliability*. Power capacitors impregnated with XFS are more reliable than the same type of capacitors impregnated with Aroclor 1016. This conclusion is based on results of comparative evaluations. The following tests have been completed. Three years of accelerated life tests, more than 18 million kilovar hours in full-sized units without failures. Hundreds of sample capacitors operating up to 200 percent of the rated voltages for a wide range of temperatures, minus 60° to plus 125° C.

6. *Economics and availability*. The long-range price of power capacitors impregnated with XFS 4169L will be reasonable. The increased price or expense attributed to the fluid is expected to be less than $20 for a 200-kilovar unit.

Dow has assured the electrical industry that it will have the manufacturing capabilities to produce XFS at a million pounds per year rate during the first quarter of 1976. Should this fluid prove to be acceptable to the industry, Dow is willing to make the commitment to produce the fluid in multimillion pound quantities per year by the end of 1976.

Health and Environmental Assessment

The Dow Chemical Company's assessment of the health and environmental acceptability of XFS 4169L as a dielectric fluid in capacitors is based on the following criteria.

1. *Biodegradability*. The components in XFS 4169L are significantly more biodegradable than the components of Aroclor 1016. With micro-organisms, the rates of formation of radioactive carbon dioxide from radiolabeled components of XFS 4169L are much faster than for 2,5,2′ trichloro biphenyl, a major component of Aroclor 1016. The major component in 4169L is 45 times more biodegradable than the representative PCB isomer. This rate of biodegradability is comparable to several nonpersistent industrial chemicals.

2. *Bioconcentration*. The bioconcentration factors in fish of the components of XFS 4169L are low relative to the components of Aroclor 1016. The environmentally significant components in each fluid show a factor 30 times less bioconcentratable in trout muscle for XFS.

3. *Toxicity to animals*. In both acute and 90-day toxicity tests with animals, XFS shows very little toxicity. For example, in rats, a dose of 10 grams per kilogram of XFS 4169L was administered orally and had no observable effect on rats.

The levels of the components of XFS that accumulated in the fat of rats fed the XFS in the diet for 150 days were significantly lower than for the comparable dietary tests with Aroclor 1016. For example, the accumulation in the fat was 22 times less for XFS; the apparent plateau level was achieved in 1 to 2 months compared to 6 to 7 months for the PCB, and the estimated half-life of the rat was 7 days with XFS compared to 60 days for the PCB. This indicates the relatively low degree of toxicity associated with XFS 4169L in animals.

4. *Toxicities to fish*. The XFS material is only moderately toxic to fish compared to capacitor-grade PCB, which is extremely toxic. The concentration of XFS that was toxic to fathead minnows was 15 milligrams per liter compared to 0.76 milligrams per liter of Aroclor 1016, which is a factor of 20 times less toxic to the fathead minnow.

Dow and McGraw-Edison Companies have determined that XFS 4169L is an acceptable alternative to capacitor-grade PCB in high-voltage power capacitors. This is based on the electrical performance and its low potential impact on health and the environment.

Today, McGraw-Edison is proceeding with field trials of capacitors at utility companies across the country. Other U.S. manufacturers of power capacitors are currently evaluating XFS for applicability in their respective companies.

The information about XFS in this discussion is only a very brief summary of the toxicological and ecological data which have been generated.

CHLORINATED BIPHENYL DIELECTRICS— THEIR UTILITY AND POTENTIAL SUBSTITUTES

David Wood*

Abstract

The paper describes the major reasons for the use of chlorinated biphenyl in capacitors and transformers. These lead to establishment of objectives for research into potential substitutes. MCS-1238, a non-PCB capacitor fluid developed by Monsanto, is discussed and areas where further development work is required are indicated. The particular problems associated with definition of "fire resistance" relative to transformer fluids is raised.

I. INTRODUCTION

In 1970, Monsanto voluntarily began its program of terminating sales of chlorinated biphenyls to open applications—those which could result in losses to the environment. By late 1972, this program was fully implemented and Monsanto was selling these products only to manufacturers of sealed electric equipment such as transformers and capacitors.

Major applications affected by our withdrawal were carbonless paper, fire-resistant hydraulic fluids, heat transfer fluids, and plasticizers. Sales for other miscellaneous minor applications were discontinued during the same period. This action resulted in a reduction of some 45 million pounds per year in the use of chlorinated biphenyl in areas where entry to the environment was less controllable.

We decided at that time to continue supply to closed electrical applications because we believed that:

1. Entry of chlorinated biphenyl to the environment was limited and controllable;
2. A more biodegradable, lower chlorinated homolog had been developed which the capacitor industry could use;
3. Withdrawal would have brought to a halt production of equipment essential to the safe and efficient distribution and use of electrical energy because there was no known satisfactory replacement for chlorinated biphenyl dielectrics.

Today we continue to sell chlorinated biphenyl, observing the following policy:

1. We supply only to manufacturers of *sealed* electrical equipment such as capacitors and transformers.
2. We supply lower chlorinated homologs, Aroclor 1016, to the capacitor industry.
3. We offer an incineration service for liquid PCB wastes.
4. We continue to work with ANSI Committee C107 and other bodies to establish appropriate handling and control procedures for equipment containing chlorinated biphenyl.
5. We allocated increased research resources in 1969 to seek and develop effective replacements; this program continues.
6. In seeking possible replacements, we will insure that differences between Aroclor and candidate fluids from our program are widely reviewed in order that the potential impact of any compromises is fully evaluated.

The implementation of these and other programs both by ourselves and electrical equipment manufacturers was prompted by the utility of this dielectric family and the difficulties inherent in developing substitutes to effectively and fully replace it.

II. UTILITY OF CHLORINATED BIPHENYL IN CAPACITORS

1. *Fire Resistance*

The adoption of chlorinated biphenyls in 1929 as capacitor dielectrics stemmed from their superior dielectric properties compared to mineral oil. However, recognition of the fire-resistant character of the fluids influenced system and equipment design and standards over the subsequent 45 years. It is probably true that today many people find it difficult to assess potential capacitor fire hazard purely because Aroclor has been used for 45 years.

Particular examples where fire resistance in a capacitor is of benefit include:

- a. fluorescent lighting ballasts,
- b. air-conditioner motor capacitors,
- c. television capacitors,
- d. large power capacitors where high fault currents can cause rupture and ejection of fluid from pole-mounted units close to people and buildings,
- e. industrial furnace capacitors.

2. *Stability*

The persistence of chlorinated biphenyl in the environment is associated with the high degree of thermal, chemical, oxidative, and hydrolytic stability which permits capacitor manufacturers to fulfill the exacting reliability requirements that exist today.

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3. *Dielectric Constant/Dielectric Strengths*

These properties are important in determining the size of a capacitor. In a mixed dielectric system, e.g., paper/Aroclor or paper/polypropylene/Aroclor, the dielectric properties of Aroclor permit optimization of stress distribution between the components making up the dielectric layer. This has enabled capacitor manufacturers to reduce paper and film volumes for a given capacitance. I shall discuss under the heading of "potential substitutes" the impact that this could have on:

a. paper/film availability and usage;
b. design of equipment containing capacitors.

III. UTILITY IN TRANSFORMERS

Chlorinated biphenyl transformers represent less than 15 percent of transformers in service. Their use is associated with the need to limit fire hazard in installations.

1. *Railroad Transformers*

Multiple unit cars as used in rapid transit systems have transformers mounted beneath each car. By nature of the type of service, involving high passenger density, safety is essential.

2. *Urban Power Substations* (e.g., Underground Vaults)

These designs need to take into account city center space limitations and also the safety of the public and maintenance crews. Fire-resistant liquid transformers are helpful to all these objectives.

3. *Industrial Load Centers*

Efficient system designs for large, power intensive, manufacturing plants (e.g., automotive assembly, steel production) often incorporate transformers close to the electrical load centers. The use of Aroclor transformers at these centers, in the heart of the plants, or overhead in roof structures, protects both employees and plant.

4. *Transformer/Rectifiers*

Programs to reduce the emission of particulate matter from stack gases, for example in fossil fuel generating plants, include installation of electrostatic precipitators. The transformer/rectifiers energizing the precipitator field must be located close to the electrodes. In many designs, the multiple transformers are located in a penthouse above the precipitator. A fire in the penthouse could lead to shutdown of the precipitator, and thus the generating plant, if pollution control is to be maintained. A fire-resistant fluid is of obvious benefit in this application.

In each of these applications, Aroclor protects the system from:

a. initiation of a transformer fluid fire by an electrical fault beneath the liquid level,
b. electrical breakdown of the fluid causing emission of flammable gases,
c. propagation of fire if the transformer liquid content is involved in an external fire.

IV. POTENTIAL SUBSTITUTES IN CAPACITORS

1. *Research Objectives*

In seeking potential substitutes, our research objectives of necessity involved keeping those properties that gave Aroclor its value. We equally recognized the need that an Aroclor replacement should eliminate environmental concerns. A replacement should ideally operate across the full range of current Aroclor capacitor applications while requiring minimum changes in design of capacitors and equipment utilizing capacitors.

The use of chlorinated biphenyl is worldwide. Monsanto manufactures chlorinated biphenyls both in America and Great Britain. We supply to the capacitor industry of many countries. We sought potential replacement products that could be made available with the consistent quality control applied to Aroclor on a worldwide basis.

We referred earlier to availability of codielectric components in capacitors. A solution requiring substantial changes in availability of polypropylene film (quantity or quality) or a major increase in short-term availability or capacitor paper, we considered unsatisfactory. If in 1974 such increased quantities had been required, they would not have been available. Capacitor production would have fallen short of demand, further jeopardizing efficient power supply.

Our research objectives can be broadly summarized in table 1.

2. *Non-PCB Candidates*

The capacitor industry is currently examining two Monsanto non-PCB (candidate) dielectrics. These contain no chlorinated biphenyl and are not chlorinated products. The two fluids are designated MCS 1238 and MCS 1588. Both of these products are blends of synthetic hydrocarbons with a high dielectric constant additive to give a dielectric constant equivalent to Aroclor 1016. Table 2 lists some of the properties of MCS 1238 and MCS 1588 compared to Aroclor 1016.

Table 1. Research objectives in seeking potential substitutes for capacitors

1. Match or exceed Aroclor 1016 capability:
   - a. Dielectric constant - Usage of other components
     Dielectric strength - Convertability
   - b. Stability - Reliability
     Power factor
   - c. Fire resistance - Safety
2. Good environmental compatibility:
3. a. Span existing applications - Complete solution.
   - b. Internationally available - Not solely U.S.A. situation.

Table 2. Some properties of MCS 1238 and MCS 1588 compared to Aroclor 1016

Property	Aroclor 1016	MCS 1238	MCS 1588
DK 25° C	5.9	6.0	6.1
100° C	4.85	5.1	5.1
Corona IV/EV[a]	-	-	-
Hydrolysis stability[a] (neutralization number)	0.00	0.00	0.00
Dissipation factor fluid tan δ 60 Hz. 100° C	0.0025	0.05	0.05
Dissipation factor model paper capacitor at 90° C	0.0032	0.0039	0.0035

[a] See text explanation, section IV 2.

Corona inception and extinction voltages are more a function of capacitor design than of the liquid itself. Preliminary industry results demonstrate functioning in capacitors equivalent to Aroclor 1016. Further full-scale work is required before final conclusions can be drawn.

Dielectric constants relate closely to those of Aroclor 1016 over the temperature range of capacitor operation. Hydrolysis stability is mentioned because of work carried out on earlier candidates based on esters, which gave concern because of hydrolysis instability. Hydrolysis was assessed by measuring the neutralization number of a sample with 0.5 percent water added, which had been heated for 168 hours at 210° F in a stainless steel bomb along with an aluminum and a mild steel coupon.

3. *Fire Resistance*

Neither MCS 1238 or 1588 is fire resistant. This deficiency versus Aroclor 1016 must be closely considered.

4. *Environmental Considerations*

The environmental/health evaluation of capacitor replacement fluids must be related to:

a. Degradation—If some quantity enters the environment, at what rate and through which mechanism will it degrade?
b. Tissue accumulation
c. Toxicity—Occupational safety
   Environmental compatability

5. *Degradation*

Biodegradation has been studied using a semicontinuous activated sludge technique. Forty-eight-hour exposure of Aroclor 1254, Aroclor 1016, and MCS 1238 dielectric fluids to activated sludge using a semicontinuous procedure resulted in the percent biodegradation rates and 95 percent confidence limits shown in table 3.

For the polychlorinated biphenyl (PCB) materials, the level of chlorination appears to be the most significant factor in their relative biodegradability. The rate of biodegradation decreases as the number of chlorine atoms per biphenyl molecule increases. Chromatograms representing samples after exposure to activated sludge show significant alteration in the Aroclor 1016 isomer distribution, but little for Aroclor 1254. Degradation of the nonhalogenated fluid, MCS 1238, proceeds much more rapidly than for the halogenated PCB fluids with no evidence of resistant components. The methodology for this technique is described in appendix B.

6. *Tissue Accumulation*

Figure 1 depicts the results of rat tissue residue level studies vs. time and compares Aroclor 1242, Aroclor 1016, and MCS 1238. A fraction of the ingested Aroclor 1242 and Aroclor 1016 was stored in rats' lipid reservoirs. However, most of this residue was depleted after the rats had been on the basal laboratory diet for several weeks. During the course of the feeding study, residues of Aroclor 1016 accumulated more slowly and to a significantly lesser extent than those of Aroclor 1242. During the recovery period, these PCB residues decreased to lower values for Aroclor 1016.

The residue concentrations of MCS 1238 quickly reached a stable level well below the concentration in the feed. The residues did not increase with continued exposure. After feeding of the treated chow was ceased, the MCS 1238 residues were rapidly metabolized and/or excreted. The tissue residue accumulation and depuration profile of MCS 1238 shown in figure 1 is markedly different than those of the Aroclor fluids, especially that of Aroclor 1242. Methodology is given in appendix A.

Table 3. Results of biodegradation using a semicontinuous activated sludge technique

Material	48-Hour percent biodegradation	Feed concentration, ppm
Aroclor 1254	15 ± 38	1
Aroclor 1016	33 ± 14	1
MCS 1238	70 ± 10	3

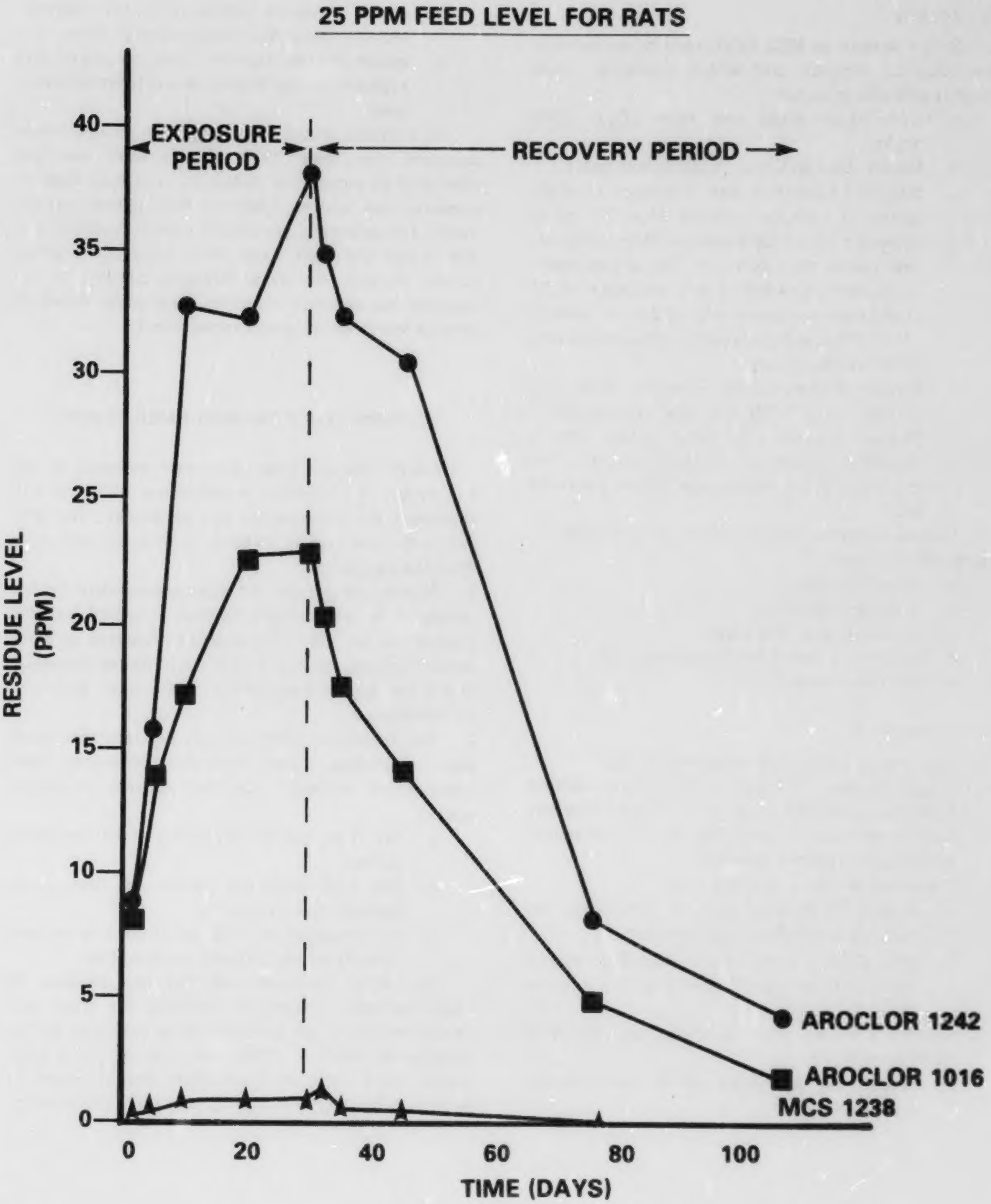

Figure 1. Results of rat tissue residue level studies vs. time, comparing Aroclor 1242, Aroclor 1016 and MCS 1238.

7. *Toxicity*

Before samples of MCS 1238 could be evaluated in the capacitor industry and within Monsanto, acute toxicity data was gathered:

- a. Rat–Acute single oral dose LD_{50}: 3,800 mg/kg.
- b. Rabbit–Dermal LD_{50}: 5,000-8,000 mg/kg.
- c. Rabbit–Potential eye irritation: A slight degree of irritation resulted when 0.1 ml of undiluted MCS 1238 was placed in the conjunctival sac of the rabbit eye. The average maximum score recorded at one and again at 24 hours after treatment was 12.0 on a scale of 110.0. All eyes had regained normal appearance 72 hours after dosing.
- d. Rabbit–Potential skin irritation: When undiluted, MCS 1238 was held in continuous 24-hour contact with intact rabbit skin, a moderate degree of irritation resulted. The maximum average score was 3.6 on a scale of 8.0.

Further programs are in process, or scheduled, to study the following:

- a. vapor inhalation,
- b. ultimate degradation,
- c. 90-day pilot feeding study,
- d. long-term (2-year) feeding studies, and
- e. fish tissue residues.

8. *Conclusions*

To summarize Monsanto research activities:

- A large number of single compounds and mixtures have been evaluated in terms of physical property data, environmental compatability, fire resistance, and model capacitor life testing.
- These have led us to conclude that:
  - a. Aroclor 1016 may well be sufficiently degradable to remain in controlled use,
  - b. MCS 1238 is a potentially acceptable replacement with the qualification that it is not fire resistant.
- Further programs must be completed with MCS 1238 in order to:
  - a. deepen our knowledge of its environmental compatibility,
  - b. permit complete evaluation by the capacitor industry across their range of applications,
  - c. enable utilities, capacitor users, and agencies to evaluate the significance of decreased fire resistance.

As a closing thought to this section, I would like to comment that since 1929 when Aroclor was first developed as a capacitor dielectric, normal commercial pressures have spurred efforts to find superior replacements. The awareness of environmental accumulation of chlorinated biphenyls from other applications added further impetus for more intensive research in the chemical and electrical industries. Aroclor has defied 45 years of search for a superior replacement.

V. SUBSTITUTE TRANSFORMER FLUIDS

Neither Monsanto nor any other company, to our knowledge, has developed a transformer dielectric with equivalent fire resistance to that of Aroclor. The difficulties that we face in common with other workers in this field are twofold:

1. Aroclor has become the reference standard for fire resistance in transformers because it works and has worked for 45 years. To establish standards to guide research effort, there is a need for objective evaluation of the fire hazard associated with the major sectors of transformer use.
2. The chemistry which imparts fire resistance tends also to produce stable molecules. Monsanto seeks replacement products that will provide protection against:
   - a. fire from transformer faults under the liquid surface,
   - b. fire from secondary ignition of gaseous arc decomposition products,
   - c. fire propagation if the transformer is involved in an externally initiated conflagration.

We strive to accomplish this and produce an environmentally compatible product. We have four candidates which are currently being evaluated by the transformer industry. These materials are in a sufficiently early stage of development that it would be premature to give detailed property data at this meeting.

APPENDIX A

METHODOLOGY FOR FEEDING STUDY

Feeding and Sampling

Rat chow containing 25 ppm Aroclor 1242, Aroclor 1016, or MCB 1238 was prepared by mixing the products into Ralston Purina rat chow. The treated chow was fed ad libitum to adult albino rats for an exposure period of 30 days. Following the 30-day exposure period, all remaining rats were placed on the basal laboratory diet. At predetermined intervals during the exposure and recovery periods, five rats from each exposed set and a control set were sacrificed. Fat tissue was excised for analysis and composited for each group. Samples were quick-frozen and stored in glass containers with aluminum foil-lined caps to minimize risk of contamination.

Isolation

The dielectric fluid residues were isolated from the fat by solvent extraction. A weighed amount of fat was placed in an Erlenmeyer flask and homogenized three times with 25 ml of pesticide-grade hexanes and anhydrous sodium sulfate using an ultrasonic homogenizer. The combined supernatants and washings were filtered through anhydrous sodium sulfate and diluted to 100 ml with hexane.

Lipid Weight Determination

A 5 ml-aliquot of the extract solution was pipetted into a tared 50-ml beaker. After evaporation of the solvent under a stream of nitrogen, the beaker and residue were reweighed to obtain the lipid weight of the aliquot. All residue levels are reported as ppm on a lipid weight basis.

PCB Cleanup And Measurement

Sample cleanup for the extracts containing Aroclor 1242 and Aroclor 1016 residues was accomplished by pipetting an aliquot of the extract onto a 5 percent deactivated alumina column and eluting with 125 ml of hexanes. The column eluate was collected in a Kuderna-Danish evaporative concentrator, a 3-ball Snyder condenser was attached, and the solution was concentrated to 5 ml. The residue levels in the extracts were measured by gas chromatography using an electron capture detector.

Non-PCB Cleanup And Measurement

Sample cleanup for the extracts containing MCS 1238 residues required separation of the residues from the lipid by preparative scale gel permeation chromatography. Following the GPC separation, the extracts were further cleaned up on an alumina column, collected, and concentrated as above. The residue levels in these extracts were measured by gas chromatography using a flame ionization detector.

Calculations

Calibration curves for each product were prepared by plotting detector response (total peak area) versus nanograms of standard injected. Residue levels in the samples were determined by summation of the total area of peaks corresponding to peaks in the standard and use of the appropriate calibration curve. The calculations were done as follows:

$$\text{Residue (ppm)} = \frac{(N)(V_F)}{(V_I)(W)}$$

where N = Amount of product from calibration curve (ng),
V_F = Volume of final concentrate (ml),
V_I = Volume injected (ul),
W = Lipid weight of original sample (g).

APPENDIX B

TECHNIQUE FOR BIODEGRADATION METHOD

Biodegradation Method

Since activated sludge is one of the most important agents for sewage treatment, test procedures evaluating its action are of great importance.

The semicontinuous activated sludge (SCAS) method has been extensively utilized in the development of biodegradable detergents. In our SCAS procedure, patterned after the Soap and Detergents Association's standard method (1,2) mixed liquor (activated sludge and supernatant) from a local domestic sewage treatment plant is charged to magnetically stirred glass vessels of 1.5-l capacity. Means for aeration and sampling are provided. The SCAS unit is generally operated using a retention or aeration time cycle of 24 to 72 hours. At the beginning of each cycle, synthetic sewage (300 mg glucose, 200 mg nutrient broth, and 130 mg K_2HPO_4) and the appropriate test material in ethanol solution are added to the mixed liquor (2,500 mg/l suspended solids concentration). Aeration is maintained until the end of the cycle, at which time the sludge is settled and 1 l of supernatant drained. The cycle is then reinitiated by the addition of tap water, synthetic sewage, and test material. Operation of the units can be continued for an indefinite period of time until consistent degradation rates are observed.

Sample Analysis

Biodegradation of the test material was determined during one cycle each week by analyzing 20 to 50 ml mixed liquor samples withdrawn after feeding and at the end of the aeration cycle. The mixed liquor analytical procedure involved extraction with three successive 25-ml portions of hexane, and drying combined extracts with anhydrous sodium sulfate. Extracts were concentrated in a Kuderna-Danish evaporative concentrator equipped with a 3-ball Snyder condenser, and measured by electron capture or flame ionization gas chromatography. Calibration curves for each product were prepared by plotting detector response (total peak area) versus nanograms of standard injected.

The percent biodegradation was calculated from the following equation:

$$\text{percent biodegradation} = (C_o - C_n)/C_o \times 100$$

where C_o and C_n use the initial and final concentration of test material, respectively, on the mixed liquor.

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SOME COMMENTS ON ALTERNATIVES TO PCB's

Bruno Rey Coquais*

Abstract

In the transformer industry, the benefit of a nonflammable liquid seems to exceed the risks of pollution by polychlorinated biphenyls; however, to minimize eventual contamination of the environment, it is suggested to use, when possible, a mixture of chlorobenzene and trichlorobiphenyl or even chlorobenzene alone.

For the impregnation of capacitors, most of the substitutes which can be imagined are not very attractive, though they are useful for certain applications. It is proposed to use a mixture of pure dichlorobiphenyls and their alkylated derivatives. This impregnant, named chloralkylene, has dielectric properties very similar to the industrial trichlorobiphenyl.

The tests in progress already show that chloralkylene is easily biodegradable and has a low toxicity. This compound seems to be quite acceptable for the environment and should be a perfect substitute for polychlorinated biphenyls in capacitors.

To begin with, I would insist on the fact that heavy chlorinated biphenyls have been used for a long time in dispersive applications as plasticizers and for other uses. Although these types of applications have been stopped in accordance with the OECD recommendations of the February 14, 1973, it is not surprising to continue to find these persistent, highly chlorinated PCB's everywhere in the environment. It is therefore very difficult to know if the electrical uses of PCB's for transformers and for capacitors have a significant contribution to the pollution of our environment.

TRANSFORMERS

PCB's are used in transformers only when the fire hazard is high (department stores, theatres, movies, skyscrapers, factories); otherwise oilfilled transformers, which are cheaper, are always preferred. We do not think dry-type transformers would be a competitive alternative in spite of the fact that silicone and epoxy belong to the range of products we market. They may be very attractive in some cases, but in addition to certain economical and technical disadvantages, dry transformers do not offer a perfectly safe solution wherever flammable vapors (solvents, oil, gas) may be present accidentally.

We believe a nonflammable dielectric liquid is absolutely necessary to prevent the risk of fire in transformers. In table 1 we have listed the different possible solutions. In the United States, at this time, transformers are filled with Inerteen 70-30, which is based on pentachlorobiphenyl, or with Inerteen 100-42. To minimize the pollution risk, a mixture of trichlorobiphenyl and chlorobenzene or even the chlorobenzene alone could be used in spite of some technological problems.

In any case, the systematic recovery of used PCB's will further limit pollution hazards; our company Prodelec has organized this practice throughout France since 1968, as soon as this pollution problem became known. This systematic recovery has now been enforced in France by the French law of July 8, 1975, on PCB's.

CAPACITORS

For capacitor impregnation, we have examined various possible solutions. We think that the performances of such compounds as silicone; sulfone or synthetic oil (alkylbenzene, polybutene); phthalate; and sebacate—which we are indeed selling ourselves for certain applications—are not really satisfactory enough for generalized or widespread use.

In our opinion, a substitute to PCB's should be characterized by:

- High permittivity;
- Nonflammability, at least not sustaining combustion;
- Compatibility with polypropylene film;
- Being a permanent liquid with no crystallization until -25°;
- Good thermal and electric subfield stability;
- Regarding its effect on the environment, being fairly easily biodegradable, to eliminate the risk of accumulation, and by having low toxicity.

To get a good and nonflammable dielectric fluid means in practice that the compound must be a chlorinated aromatic hydrocarbon. We have studied, for instance, new structures, as shown in figure 1. With chlorobiphenyl oxide, we are afraid of the risk of metabolization in highly toxic dioxins and we feel that products of the family of the chlorobiphenylethane are technically and environmentally much more promising. Actually, introducing a totally new compound would require a long and expensive testing program and it seems better to take advantage of the huge amount of knowledge gathered about PCB's and to see how it may be possible to improve the present situation.

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Table 1. Possible solutions for dielectric liquid

IEC type	Chlorobiphenyl	Chlorobenzene	Pour Point (°C)	Viscosity Cst at 20° C	ε at 20° C	Hydrogen index[a]	Askarels for transformer
T_1	60% hexa	40% tri	<-33	21	4.5	<0 excess Cl	Pyralene T_1 Pyroclor
T_2	45% hexa	55% tri and tetra	<-40	11	5	<0 excess Cl	
T_4	70% penta	30% tri	<-30	20	5	0	Inerteen 70-30 Pyralene T_4
	45% penta	55% tri and tetra	<-40	10		<0 excess Cl	Pyralene T_2
T_3	100% tri		<-18	65	6	16	Pyralene T_3 Inerteen 100-42
	60% tri	40% tri	<-50	7	6	10	Pyralene 1460
	40% tri	60% tri and tetra	<-20			4	
		100% tri and tetra	Chrystallize -9		6	<0 excess Cl	

[a] Hydrogen index = $\dfrac{\text{No. hydrogen - No. chlorine}}{\text{Molecular weight}}$.

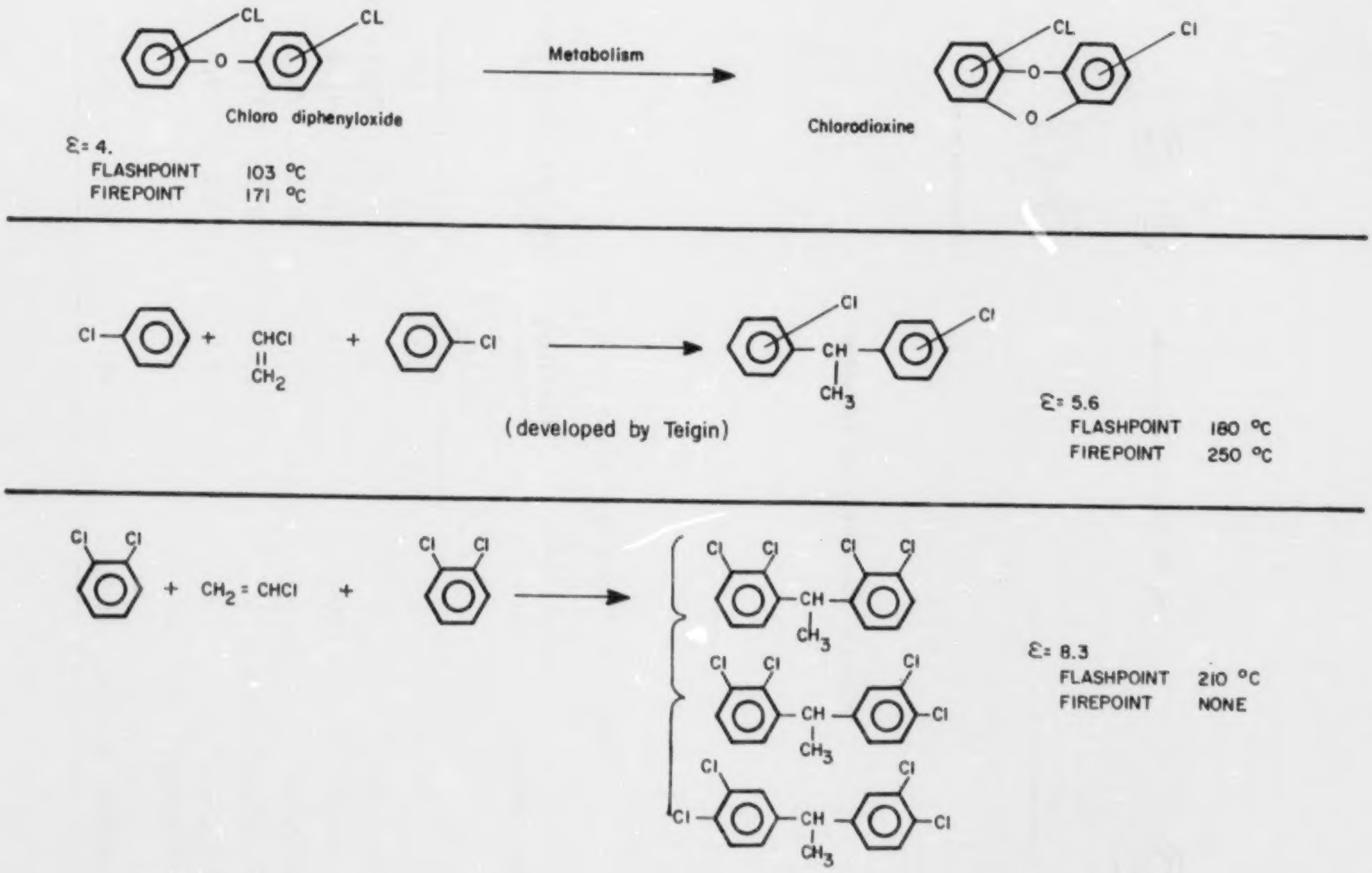

Figure 1. New structures for nonflammable dielectric fluids.

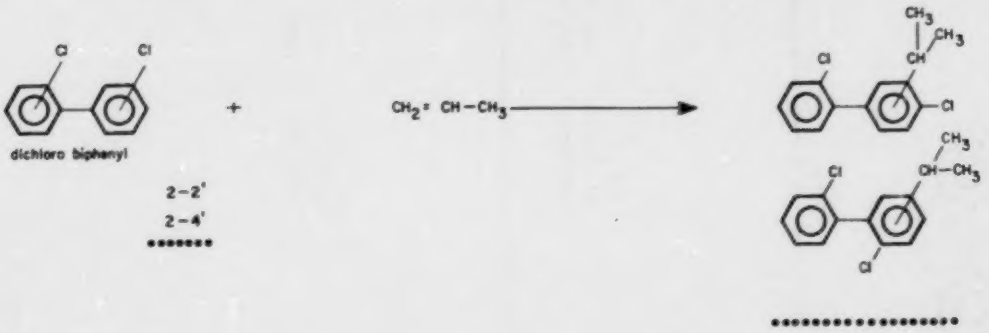

Figure 2. Structure of chloralkylene.

Table 2. Composition of various impregnants

Composition, percent	Chloralkylene 12	Pyralene 3010 (trichloro-biphenyl)	Pyralene 1500	Pyralene 2000
Biphenyl	0	0	0	1.6
Monochlorobiphenyl	0	0	0.5	18.4
Dichlorobiphenyl	20	11.4	37.5	44.0
Trichlorobiphenyl	0	57.1	40.0	23.4
Tetrachlorobiphenyl	0	29.4	20.5	11.7
Pentachlorobiphenyl	0	2.2	1.5	0.9
Alkylchlorobiphenyl isomers	80	-	-	-
Chlorine content, percent	25.8	42.	38.5	33.5

Table 3. Comparison of chloralkylene to commercial trichlorobiphenyls

Characteristics	Chloralkylene 12	Pyralene 3010 (trichlorodiphenyl)
Specific gravity at 20° C	1.163	1.391
100° C	1.097	1.319
Coefficient of expansion	7.5×10^{-4}	6.8×10^{-4}
Viscosity Cst. at 20° C	135	65
100° C	3.2	2.3
Pour point (ASTM D 97)	-25° C	-23° C
Firepoint (Cleveland)	258° C	none until boiling
Permitivity at 20° C	6.00	5.93
100° C	4.86	4.80
Resistivity 100° C - 500 V - 1 mn	$>3000 \times 10^9$	$>3000 \times 10^9$
Dissipation factor +gδ 100° C - 50 cps	<0.02	<0.02

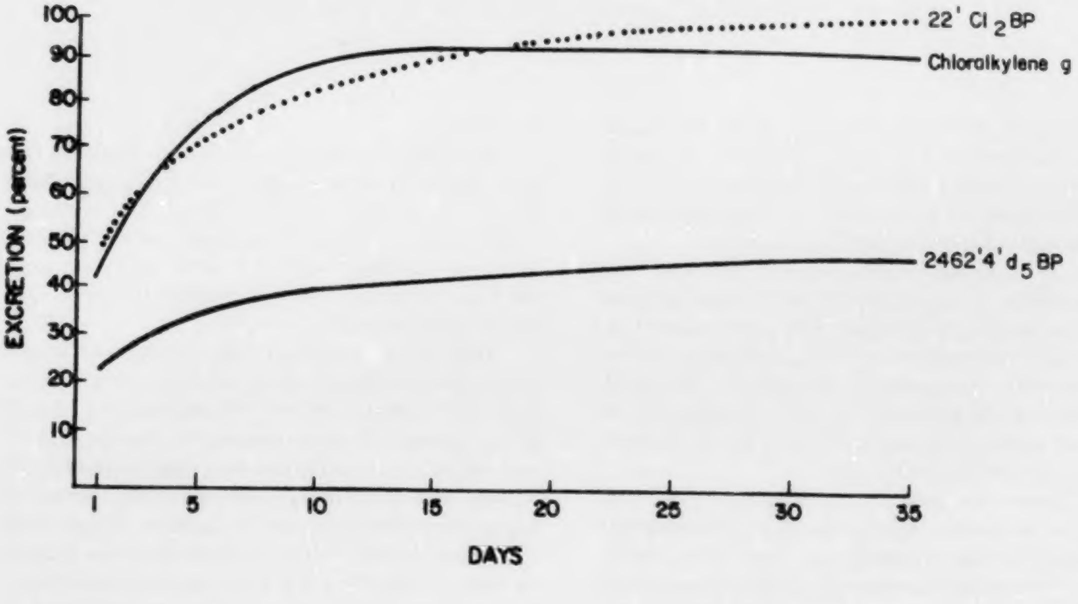

Figure 3. Excretion by rats, with diet of 2 ppm/day for 5 weeks.

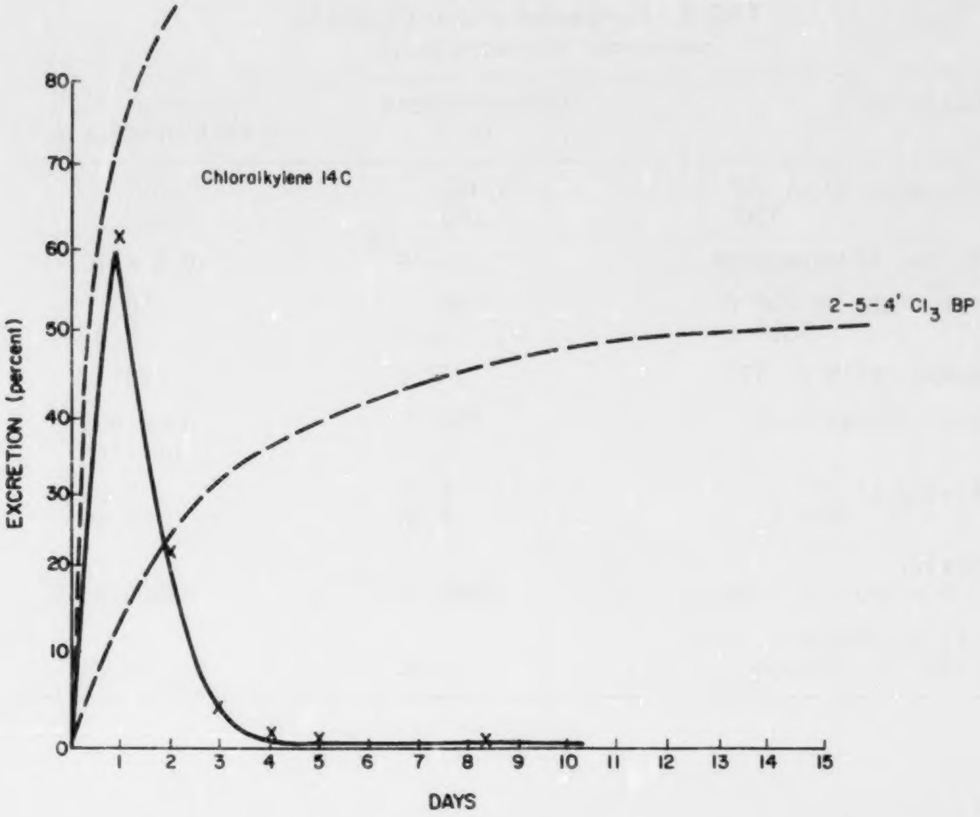

Figure 4. Excretion by rhesus monkey; single oral dose 1 mg/kg.

The studies carried on, in particular by Prof. Korte and Dr. Klein in Germany, seem to prove the easy biodegradability of PCB's when they have a very low chlorine content; therefore, pure mono- or dichlorobiphenyls could be quite acceptable for the environment.

As an impregnant consisting only of mono- and dichlorobiphenyls would crystallize at room temperature, it is necessary to produce a more complicated mixture to get a permanent liquid at all temperatures. We have therefore developed a product named "chloralkylene," obtained by addition of an alkyl chain, actually an isopropyl group, on a mixture of nearly pure dichlorobiphenyl isomers (figure 2).

As shown on table 2, chloralkylene contains no penta- nor tetrachlorobiphenyl and the trichlorobiphenyl content can be extremely low. Technically, for its dielectric and physical properties, the chloralkylene is a perfect substitute to the commercial trichlorobiphenyls (Pyralene 3010, Arochlor 1016), as shown in table 3 and as checked by some major capacitor manufacturers here and abroad.

To assess its impact on the environment, we have given chloralkylene to the Institute for Ecological Chemistry in Bonn, Germany, Prof. Korte. This institute is engaged in an extensive investigation on "PCB's in the Environment." We therefore hope to have next spring the final results on the fate of chloralkylene in comparison with PCB isomers.

Tests on environmental acceptability are actually being carried out with chloralkylene based on 2-4′ dichlorobiphenyl labeled with 14c. The tests consist of a study of the balance of the excretion, the storage, and the metabolism in rats and in monkeys (figures 3 and 4); the monkey studies are being completed at the Institute of Experimental Pathology and Toxicology, Albany Medical College, Albany, New York. These tests also examine the fate of chloralkylene in a soil plant ecosystem and in an aquatic ecosystem, as well as its behavior under atmospheric conditions, upon waste composting, and in sewage treatments. The results so far obtained prove that chloralkylene does not accumulate, is metabolized quickly, and behaves generally like the dichlorobiphenyls isomers themselves.

Chloralkylene is two times less toxic than trichlorobiphenyl when one compares the acute toxicity values. At this time, there is no real reason to consider PCB's dangerous for the labor manufacturing capacitors, if the necessary precautions are taken. We think that with an easily biodegradable compound such as chloralkylene, long-term effects due to accumulation along the food chain do not need to be feared.

The chloralkylene price is presently twice that of the currently commercially available polychlorobiphenyls, which would result in an increase of the capacitor costs of about 5/10 percent. However, by substituting mineral oil or other substitutes with less outstanding characteristics, the increase of the cost of capacitors would be much higher. We hope to improve its economy and are quite confident that chloralkylene could thus be an excellent substitute for trichlorobiphenyls.

PCB's AND THEIR SUBSTITUTES – A BRIEF LOOK AT SOME EXAMPLES OF PAST TRADEOFFS

Dale Hattis, Ph.D., and Albert Murray, Ph.D.*

Abstract

Some aspects are described of a study of the economic, health, legal, and other impacts of the past voluntary restriction on PCB sales. Examples illustrate the importance of examining the effects of substitute technologies in assessing regulatory policy by showing the potential for surprises in this regard. In the PCB case, substitutes may be producing both unanticipated economic benefits and unappreciated health and safety risks.

I am Dale Hattis of the Center for Policy Alternatives at M.I.T. My colleague at the center, Dr. Albert Murray, who was originally scheduled to speak here—and who would be the most appropriate speaker on this subject—was unfortunately taken ill about a week and a half ago and he is now still recuperating in the hospital. What I have done in his place for presentation today is to extract some particular examples of tradeoffs from Al's work on PCB's, which illustrate some occasionally surprising features of the technological changes that arise out of environmental/health concerns.

For context, I would like to say that our project, sponsored by the Council on Environmental Quality and EPA, and under the direction of Dr. Nicholas A. Ashford, has for the past year been exploring ways to analyze the economic, environmental health, legal, and other impacts of regulatory decisions related to environmental chemicals. In this exploration, we are now in the process of completing eight experiments in the analysis of the impacts of particular regulatory actions, primarily drawn from the past 5 years of history. The first of our eight experiments, though not strictly resulting from a mandatory governmental action, was the restriction a few years ago of the sale of PCB's for particular uses. Because our case studies are experiments to explore the practical use of methods, and because the resources we could devote to each case were relatively small, we could not perform as comprehensive an elucidation of effects as we would have wished. Nevertheless, we do think that our analyses have revealed important types of questions, similar to those referred to yesterday by Mr. Train, which are difficult to deal with in the single chemical reactive context of most current regulatory authorities.

My initial two examples of such questions on PCB's illustrate the fact that it is sometimes difficult to be sure that technological changes introduced with the intent of reducing environmental health risks accomplish their purpose on balance, when the risks of substitute practices are considered. Sometimes there appear to be legitimate reasons for concern that particular changes—especially in the absence of continuing regulatory followup—might create environmental health hazards as dangerous or more dangerous than the ones that are eliminated.

As a first example, the substitutes for PCB's in some large heat exchangers and in some special hydraulic fluids for high-fire-risk uses are flammable at elevated temperatures. Concern for the lack of fire protection previously provided by PCB's in these situations has reportedly caused a substantial increase, as much as a doubling in some cases, in fire insurance rates on some industrial facilities employing high-temperature heat exchangers. In our case study, Al Murray estimated the total increase in premiums at possibly in the tens of millions of dollars annually. This, of course, reflects mainly property damage and not the human cost. However, if our information on the increased premiums is correct, and if the apparent perception of the insurance companies is correct as to the size of the added chance of catastrophic fires, then we must consider that such events would be likely to be accompanied by appreciable human casualties. The rise in insurance rates, of course, may not reflect an actual change in risk, and in that case the increased insurance cost is simply a transfer payment to the benefit of the insurance companies, but the matter seems to deserve further exploration with the aid of hard statistics on actual fire-risk experience. If the data confirm that appreciable human impact may be expected to occur, alternative means for reducing this risk might be productively evaluated.

Another, although probably less important, example of potential risk of substitute technology arises in the case of the former use of PCB's as a dye-solvent for carbonless carbon paper. It may be recalled that at some frequency PCB's from this source found their way into recycled paperboard products for food contact use, and this was a source of concern for the FDA. Now the PCB's formerly used for this purpose have been replaced, but unfortunately it is not public information what exactly has replaced them. The barrier of trade secrecy in this instance, as in many others, can frustrate attempts to assess the difference in environmental health risk produced by the change in technology.

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Two other examples illustrate the potential for a different category of surprises–those in the economic area.

Formerly, PCB's costing on the order of 25 cents per pound were used in many paints and coatings, sometimes as a major ingredient. In this application PCB's were a chemically stable blender and conferred resistance to fire, impacts, water, and weathering. Our inquiries with paint manufacturers indicate that chlorinated paraffins now perform essentially all the technical functions of PCB's at about half the former cost. Our industry informants mention some increased problems in a few of their products with lowered stability to dechlorination–causing occasional discoloration and loss of weathering resistance–but over the great bulk of their product lines, the industry perception appears to be that the change has been economically beneficial. PCB's had evidently become established as paint additives before the availability and advantages of the chlorinated paraffins became widely appreciated. Later, when approximate technical equality and lower prices might have tempted paint manufacturers to substitute, the usual inertial resistance to any change appears to have caused this opportunity to be generally neglected. The real world and the economists' ideal one occasionally behave differently.

Another example of a surprise (at least to us) in the economic area is the potential importance of even small adverse impacts on sport fishing. A U.S. Department of Interior survey (ref. 1) indicates that in 1970 a total of about 700 million person-days and five billion dollars were spent in pursuit of this pastime – mainly in fresh water. The five billion dollars must, of course, be considered an underestimate of the actual "value" of that activity to people. If PCB's had continued to be used as they were in the past, we might speculate that additional concern of people about residues (such as that now publicly in evidence), or possibly some declines in fish populations might have led to small fractional declines in participation or enjoyment of fishing by some portion of the population. If this were to occur, the loss of value to society of even less than a fraction of a percent of national sport fishing activities might be appraised at tens of millions of dollars annually–depending on the "value" realized by former fishers in substitute recreational activities.

The importance of examining subtle and uncertain effects, including the effects of substitute technologies, is illustrated by such occasional insights into their potential magnitude.

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ENJ-2065—AN ELECTRICAL INSULATING FLUID

E. J. Inchalik, Ph.D.*

Abstract

The need for an electrical insulating fluid whose use would avoid the adverse toxicological and ecological effects associated with polychlorinated biphenyls prompted a study of organic esters as possible substitutes. The study culminated in the identification of diisononyl phthalate (ENJ-2065) as a dielectric fluid of potential interest to the capacitor industry. ENJ-2065 has been made available to that industry and it is now being used in some commercial capacitors.

In late 1970, a study was begun at Exxon Research and Engineering Company by Drs. A. J. Rutkowski and E. O. Forster to develop an electrical insulating fluid whose use would avoid the adverse toxicological and ecological effects associated with polychlorinated biphenyls. This study culminated in the identification of diisononyl phthalate (ENJ-2065) as a dielectric fluid of potential interest to the capacitor industry. Exxon Chemical Company U.S.A. has made ENJ-2065 available to the industry for its evaluation and it is now being used in some commercial capacitors.

In the preliminary phases of this study, the dielectric properties of a significant number of mono- and dibasic acid esters were determined in order to obtain a general understanding of the interrelation between molecular structure and dielectric properties. From these data it was concluded that major attention ought to be directed at the esters of phthalic acid. The phthalates that we tested all had dielectric constants of 4-6, close enough to the desired level for fluids for the widely used paper-based capacitors to justify further evaluation. The optimum dielectric constant for these capacitor fluids is one which closely matches the dielectric constant of the paper. A close match reduces electric field inhomogeneities, increases dielectric strength and lifetime, and decreases capacitor size. Other important property criteria for a paper dielectric fluid include a low dissipation factor, to reduce energy loss and destructive heat buildup; a high dielectric strength, to reduce capacitor size and improve service life by permitting short-term exposure of the fluid to abnormally high stresses without breakdown; a low gassing tendency, to avoid production of gases that could lead to pressure buildup in sealed units; stability at elevated temperatures, to prevent capacitor breakdown and stabilize performance; low viscosity, to allow for easier impregnation and filling of capacitors and elimination of air pockets; a low order of toxicity; and compatibility with the environment.

Consideration of these factors as well as availability and cost resulted in narrowing the choice of potential candidates to those shown in table 1. From this list, diisononyl phthalate (ENJ-2065) was selected as the most promising for additional study. ENJ-2065 was chosen on the basis that it has (1) a higher dielectric constant than the phthalates of higher molecular weight, (2) an advantage in its loss characteristics relative to the lower molecular weight dihexyl and dioctyl phthalates, (3) a relatively high flash point of 430° F, (4) a more highly branched molecular structure than dihexyl and dioctyl phthalates with the potential for improved hydrolytic stability and (5) a good balance of other physical and electrical properties. ENJ-2065 is manufactured in the United States by Exxon Chemical Company U.S.A. from phthalic anhydride and a mixture of branched isomeric alcohols in which C_9 alcohols predominate.

Electrical insulating fluids have to remain essentially unchanged chemically when subjected to temperature cycles. Once having chosen ENJ-2065 for further evaluation, it was desirable to know what effect certain contaminants might have on its stability, how these contaminants might be removed, and what additives, if any, might be useful in enhancing its stability.

The effects of small amounts of two potential contaminants, alcohol and water, on conductivity of ENJ-2065 are shown in tables 2 and 3. The results show that alcohol levels of less than about 1,000 ppm and water levels of less than about 100 ppm can probably be tolerated without affecting performance seriously. To remove small quantities of these impurities as well as any acids or catalyst residues from the production of ENJ-2065, we found percolation through a packed column of activated Attapulgus clay to be effective, but we did not carry out extensive studies to optimize a purification system. It is our feeling that systems now being used in the industry for purification of PCB's prior to use in capacitors, or slight modifications of them, will prove to be satisfactory for ENJ-2065.

Although the most widely used capacitors today are of the all-paper type, newer types based on polypropylene film and paper or on all-polypropylene film are growing in importance. With capacitors of this type, the dielectric constant of the fluid is less important.

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Table 1. Electrical and physical properties of phthalate esters

Phthalate Ester	Dihexyl	Di-2-ethylhexyl	Diisooctyl	ENJ-2065	Diisodecyl	Ditridecyl
Dielectric constant	5.64	5.33	4.97	4.66	4.45	4.08
Tan delta	--	0.14	0.30	0.05	0.02	0.01
AC conductivity, 10^{-11} $(\text{ohm-cm})^{-1}$	9.0	2.1	1.3	0.29	0.20	0.04
Breakdown voltage (KV/0.1 in.)	--	28	27	30	36	29
Boiling point--mid @ 5 mmHg, °C	210	230	235	252	256	286
Pour point, °C	-33	-50	-45	-48	-50	-37
Viscosity, cps., 20° C	50	81	83	95	110	230
Flashpoint, COC, °F	380	425	430	430	452	470
Firepoint, COC, °F	420	475	485	495	515	555

Table 2. Effect of residual alcohol on conductivity of ENJ-2065

Temperature cycle (1,000 V, 60 Hz)	Conductivity $(10^{-12}$ ohm-cm$)^{-1}$		
	Wt. percent alcohol added		
	0	0.01	0.1
25° C	0.78	1.97	1.98
90° C	40	65	320
25° C	3	4	12

Table 3. Effect of residual water on conductivity of ENJ-2065

Temperature cycle (1,000 V, 60 Hz)	Conductivity $(10^{-12}$ ohm-cm$)^{-1}$			
	Wt. percent water added			
	0	0.001	0.01	0.1
25° C	0.78	2.7	3.7	135
90° C	40	165	>400	>400
25° C	3	10	25	70

Table 4. Weight gain of PP film in contact with dielectric fluid

Fluid	Run number		
	1	2	3
ENJ-2065	11.0	10.9	13.8
Arochlor 1242	18.8	18.2	22.8

Impregnation of the polypropylene film with the fluid, however, is of great importance and prompted a brief study, summarized on table 4. These data show that ENJ-2065 swells polypropylene capacitor-grade films satisfactorily, as measured by the film weight gain after 2 days at 60° C, so that film impregnation should not be a serious problem. The next generation of capacitors may well be based on metallized polypropylene film. Polychlorinated biphenyls cannot be used for this type of capacitor since they generate hydrogen chloride, which can lead to premature breakdown.

In summary, we believe that ENJ-2065 has a balance of properties such that it will find a niche in the capacitor fluid field.

GENERAL DISCUSSION OF SESSION V

CHAIRMAN MUIR: All right. I see that we have reached our scheduled departure hour. I think it's only fair to entertain a few questions from the audience and given the diversity, I would request that people raise a question from the floor and then address their questions to the individual they would like an answer from.

MR. BEN KININGHAM (Illinois Lung Association, Springfield, Illinois): I do not have any particular gentleman on the panel to address. I just want to refer to the breakdown of the film, if anyone could elaborate on how that comes about, and if so, is there any research that is being carried on now in development of films that would be any more stable.

MR. RICHARD ROLLINS (Jard Corporation, Bennington, Vermont): Are you talking about a biological breakdown?

MR. KININGHAM: Biological or chemical or both breakdowns.

MR. ROLLINS: It is a reduction type atmosphere in the capacitor so it does not see the oxygen that would normally be considered. The basic problem is not one of the degradation as much as it is an inherent characteristic of not being able to survive voltage levels or stresses in an AC application.

MR. KININGHAM: Would it be feasible to develop an alternative program?

MR. ROLLINS: The organic type films have been used or have been attempted to be used in AC capacitors. The basic problem is one of corona exception and corona distinction voltages, and this is inherently low and goes down to initiation of points 250 to 275 volts and the extinction voltage is extremely low. The meaning of the corona is that the capacitor slowly deteriorates by this ionic device which is the definition of corona. The interpretations of corona I am not going to get into today because every capacitor designer has his own opinion, but every capacitor designer is completely aware of what the corona problems can do. So essentially what we are saying is that organic films have the basic problem of not being able to withstand high AC voltages, especially in the dry system.

On an impregnated system, the circumstances are still there, so you are not really changing the circumstances a great deal in all polypropylene film or any organic film when you impregnate it.

VOICE: I have a question for Dave Wood. We heard a number of things about environmental factors. How about a still lower chlorinated material than 1016 that might be a more desirable material? I believe Monsanto has passed a given developmental material such as 1043, which is a lower chlorinated material.

MR. DAVID WOOD (Monsanto Industrial Chemicals Company, St. Louis, Missouri): We have done considerable work with MCS 1043, but at the present time commercialization of such a product must hang in the balance pending further conclusions being drawn about the true significance in the differences observed between the trichlorinated materials and bichlorinated materials. Aroclor 1016 was itself a reduction in materials from the Aroclor 1042 widely used by the industry.

In order to carry through development of the dichlorinated material, I think we need some approvals from the community that they recognize a movement in that direction as one that should be taken.

So yes, some data are already available. Acceleration or stopping of that program depends largely, on some of the things that we hope will come out of this conference this week.

MS. NANCY STROUP (Environmental Defense Foundation, Washington, D.C.): I apologize for the openhandedness of my question, but I hope that the panel members will respond as concisely as possible.

One of the speakers mentioned that his concern was that we not jump from the frying pan into the fire, and I am sure it's the concern of everyone at this meeting. To that end I would like to know when the four or five compounds that are now being developed to replace PCB's will be tested for the health and safety of these compounds, including carcinogenic, immunogenic, and teratogenic tests before your marketing. And whether this information will be available for independent review.

DR. MUIR: Essentially, as I already outlined in my talk, we developed a date for the work and the work is going forward and we recognize the biphenyl problem and that replacing the product inevitably is going to have to satisfy the most thorough and rigorous testing.

DR. DEAN BRANSON (Dow Chemical, Inc., Midland, Michigan): In the case of methosilicones additional data will be published very shortly and will be publicly available. I think this is most in the minds of all manufacturers when they consider substitutes for the material which is dielectrically one of the best ever developed. This will seem to solve the problem when it comes out.

DR. E. J. INCHALIK (Exxon Chemical Company, Linden, New Jersey): A great deal of information is already available. You may recall in 1972 a program sponsored by the National Foundation of Health Sciences and one subject made by Dr. Tapper, what he observed at comparison of PCB and mercury, it was like an ideology searching for a disease, and that's a quote.

The recent study by Dr. Hartung of the University of Michigan has concluded that no problems attributable to polyesters have been noted due to the constant low level exposure of the general population, and that occupationally, only mild skin irritation has been observed at high exposures. But he did point out that ecological work should be continued. And I might add that Dr. Tapper suggested the same.

To help answer these questions, particularly related to these items, a research grant has been made by the University of Missouri to the Manufacturing Chemists Association, and they are starting tests. I think we can hope within the next year or so results will be coming from this study obviously available to all which will help answer these questions.

MR. DUNCAN MacARUTHUR (Booze Allen and Hamilton, Inc., New York, New York): Well, some of the things that you have asked for are in progress and when these things are available, they will also be made available to the general public.

MR. DAVID C. MORRIS (Weyerhauser Company, Tacoma, Washington): You stated, sir, that a cost of $4.9 million would be necessary for I guess you would call them secondary market users like my company, to replace our PCB with some substitute.

Did I understand you correctly, Mr. MacArthur?

MR. MacARTHUR: Yes.

MR. MOSS: Just for my own sake, I just believe in it very strongly, the cost alone is estimated at $2 million and we are right in the midst of investing. And I think somewhere along the line the investing might not take into account the factors that we initially have to face. I would guess it's at least 50 times over.

CHAIRMAN MUIR: Yes, sir.

VOICE: There are three experimental items coming on the market. Will someone label these transformers--

MR. ROLLINS: Well, I'm not too sure, but there have been requests that major capacitors manufacturers now conform by labeling all capacitors that have greater than I believe 5 pounds of Aroclor—I may be wrong, but on small capacitors, the basic problem is the label gets hidden because this is placed into another piece of equipment.

MR. STEVE NUMUS (Environmental Protection Agency): I have a question for Richard Montgomery regarding the use of silicone which was approved by Underwriters Lab for indoor use. Do you know the reasons why it was approved for indoor use and why it was not approved for outdoor use?

And secondly, can somebody give me an idea of the percent of transformers in the United States used for outdoor purposes as opposed to indoor purposes?

MR. RICHARD H. MONTGOMERY (Dow Corning Corporation, Midland, Michigan): First of all, Underwriters Laboratory does not approve of any particular material for any given use—once you exceed 600 volts in a transformer, Underwriter's Laboratories has no certification. The only certification received from Underwriters' Laboratories is as to the fire hazard of your particular material on a scale which ranges from 0 to 100, with the burnability of water as 0, and gasoline as 100. Therefore, Underwriter's Laboratory does not approve silicone material for indoor use.

In respect to the use of dielectric fluids in transformers, the electric code for outdoor uses does not specify nonflammable material. It does in subsection 450, paragraph 23 approve the use for the indoors. These are the only materials approved for use.

MR. NUMUS: I want to know the percentage as opposed to indoor use.

MR. MONTGOMERY: Well, my research seems to indicate 85 to 90 percent of the transformers sold in the United States are currently outdoor applications, and the other 10 to 15 percent are indoor applications, for safety is of paramount importance.

MR. WOOD: Essentially the figure that he is using is pretty accurate. Very large power transformers are filled with mineral oil because, if they're out in the field, there is not a substantial hazard, they can be screened out. In the medium voltage range the use of askarel is something under 15 percent of the transformers used in that medium voltage area.

MR. CLIFFORD H. TUTTLE (Aerovox Industries, Inc., New Bedford, Massachusetts): I really have two questions—another alternative mentioned up there, the capacitor for getting the high voltage can consume 20 to 50 million pounds a year. How long would it take fluid to be available in that type of quantity; 1 year, 2 years, 3 years, or what? I don't want to pressure you, I just want to determine.

The second question is a third of that market is lighting, fluorescence. If you have an application at 100° C, are any of these fluids or alternatives capable of operating at that temperature without breaking?

MR. MONTGOMERY: Let me answer your second question first. The answer is yes. The answer to the first question is we do have the productive capabilities capable to handle that market. It could be accomplished very quickly. It would probably be faster than the industry takes to evaluate and qualify new material.

MR. WOOD: In terms of Monsanto, alternatives could be made available on a production basis in calendar year 1976, in relationship to the stability of these materials at 100° in the HIB and the fluorescent lighting capacitors, these are test sequences which are going on under our test programs. And the results will be forthcoming. We are in a period now of collective work. We are not ready to go tomorrow.

20 November 1975

Session VI:

GENERAL SESSION

Christopher M. Timm[*]
Session Chairman

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OPEN DISCUSSION

DR. JOHN BUCKLEY (Environmental Protection Agency, Washington, D.C.): I'd like to call to order the evening session which is our General Session. There are some papers to be presented tonight that are technical papers appropriate to other parts of the session but which just didn't fit in. In the rest of the sessions we've tried to avoid people expressing their opinions. So with that, I'd like to turn it over to our Chairman for the evening, Chris Timm, who is the Director of the Surveillance and Analysis Division for EPA here in Chicago.

MR. CHRISTOPHER TIMM (Environmental Protection Agency, Chicago, Illinois): Thank you, John. I have at the present time 26 registered speakers. There won't be any discussion during this session.

I'd like to start off by introducing the groups that have been most directly impacted by PCB's in the environment, at least as far as their livelihoods. They are the real reason we have conferences like this. We have a group of commercial fishermen, commercial as far as both the market and as far as recreational aspects in the charter fishing, who are here from Lake Michigan and some of the surrounding areas. I will lead off with them. I'll mix it up after that with technical presentations, industrial viewpoints, environmental groups, etc.

MS. JEAN HERMES (Spokesperson for Commercial Fishing Interests, Green Bay, Wisconsin): I would like you to know that hardships are already being inflicted on families because of the PCB poisoning of our waters. We are fourth-generation commercial fishermen out of Green Bay. We have four children and have had our livelihood taken away because of high levels of PCB's in the Green Bay waters.

On the last part of July of this year, the FDA shut down our business when fish tested from our nets measured 54 ppm of PCB's. Overnight we've lost a business it's taken 15 years to build.

As PCB's are almost indestructible, any further discharge, no matter how small, would only add to an already insurmountable problem. The mills dumping PCB's into the waters have gotten rich at everyone's expense but their own. It is time they make a few sacrifices. Industries have been allowed to pollute and poison the waters long enough. Every commercial fisherman in Wisconsin will soon face our situation.

The commercial fisherman provides the public with a food that is 75 percent protein. In these days of food shortages, it is a crime to let this commodity be destroyed. But the commercial fisherman will not suffer alone. Sportfishing, all water recreation, and tourist towns are threatened. Even our drinking water will become, if it is not already, hazardous. Someone must be held responsible for stopping PCB discharge into our waters before they are destroyed beyond repair. Thank you.

MS. GLORIANNE HERMES (Spokesperson for Commercial Fishing Interests, Green Bay, Wisconsin): We are from another family with seven children that fishes carp commercially from the waters of Green Bay. We have also lost our livelihood because of the high levels of PCB in carp taken from Green Bay waters. Our fish measured up to 57 ppm when tested by the Agriculture Department and we were forced to release the 15,000 pounds of carp that were in our holding ponds at that time.

The PCB problem so far has been handled backwards. My husband and his brothers were stopped from fishing carp on Green Bay, while nothing has been done to stop the sources of PCB's. The fisherman who had no part in the PCB pollution has been inflicted with great hardships after many years of building his business.

The innocent have been made to pay the price for the destruction caused by a few industries that are getting richer everyday and do not want to reinvest a small part of their profits into the treatment of their waste.

We believed that the EPA and DNR were keeping the waters clean and safe, which is supposed to be their job. How has our water been allowed to become so chemically polluted as to make its fish inedible even for animal consumption or even for use as a fertilizer? Some people have not been doing their job. For if the industries' discharges were safe, so would the water and fish be safe.

The DNR still plans to plant trout and salmon into the waters although the levels of PCB in these fish tested out even higher than some of our carp. Also, they claim that no money is available to compensate commercial fishermen for their losses due to the PCB problem when they still plan on planting trout and salmon into the polluted water.

It isn't going to help to lower the limit of PCB's

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from 5 ppm to 2 ppm, as nothing has been done to stop the dumping of PCB's even at the 5-ppm level. To lower the limit of PCB's in fish from 5 ppm to 2 ppm would destroy the fishing industry all over the United States. The levels of PCB in all other edible products, such as meat, would also have to be lowered and sport fishing would have to be stopped altogether. The fishermen would have to be compensated for their losses, which resulted from other parties' ruthlessness and carelessness.

Please attack this problem where it started by stopping all PCB discharges into the water immediately. Thank you.

MR. GENE LAMBRICH (Spokesperson for Commercial Fishing Interests, Green Bay, Wisconsin, and representative of Lambrich Brothers Live Fish Company): We haul fish from the Hermes Brothers and I think something that a lot of people don't realize is that carp is a good fish. It's just not a scavenger, it's something that's a necessity. We have two fishing resorts in St. Louis, Missouri. It has cost us roughly over 100 thousand dollars since July of this year and I think that our load was a sample load that the Federal Food and Drug Administration took the samples off of and said they were polluted.

Now if these fish were polluted at the time we inquired, in February 1975, they did state that there were PCB's in the water; why weren't they periodically checked and sampled, and if there was pollution at that time, why wasn't it stopped? We're just one of a few fish haulers. This goes all over the United States. For the life of me I can't figure out why they cannot do something with the people putting these pollutions in the waters. Thank you.

LEE WEDDIG (National Fisheries Institute, Washington, D.C.): We're a trade association consisting of 550 companies engaged in the commercial fish and seafood business.

This is somewhat of an old refrain here. In the last several years we've talked about things like DDT in our waters, which had its toll. After that it was mercury, and today it's PCB. In each of these situations, there was a cost to our business, which is a 6 billion dollar industry in the United States. It represents the livelihood of 140 thousand people throughout the entire country. In each of these situations, our industry faces three losses. The first is the direct loss of income, such as has been related to you by the people who were up here a few minutes ago, as their products become unsaleable. Then we have the loss that is harder to define yet was alluded to today, and that's the destruction of habitat for our resource. Many of our resources are failing. We're running out of fish, there's been speculation that perhaps we're overfishing. But as we learn what these chemicals do to the ability of fish to reproduce, we would have to believe that the real cause of some of the failures in our resources is because of the pollution that has been caused by the unchecked dumping of chemicals into the water.

And then finally we have a loss of consumer confidence. People don't understand that in a case of PCB's, there are certain fish that are taken from the market because they do exceed the Food and Drug Administration tolerances. The consumer doesn't really understand that this affects only a very small portion of the total and the reaction is—Well, let's not use any fish—which is certainly an unjustifiable attitude, but nonetheless, it does exist.

It's also very easy for one to say the percentage of loss is small. We are a 6 billion dollar industry; we supply something like 10 billion pounds of fish a year to the consumer. One can listen to the folks from Green Bay and say, "Well, perhaps you could lose several hundred thousand pounds of fish a year and it doesn't really amount to much in the way of percentage." But yet for these folks, it's 100 percent.

Those are the losses that our industry has. What we're recommending to this group to do, is to muster its strength, to come up with six different points. We have to solve the problem. We cannot just live with the DDT's, the mercury, the PCB's, or whatever it will be next year. We have to come to a solution. So we're recommending six things.

First for the immediate. The Government should immediately ban further sale, production, importation, or recycling of PCB's in any form, with perhaps an exception for use in existing electrical transformers where usage should be strictly controlled. But after hearing this afternoon's statement about possible substitutes that have been used in other countries, I'm not too sure I even agree to my exception any more.

Efforts should be made to achieve an international agreement to the same goals. If legislation is necessary to achieve such a ban, then we should all get together to get it passed.

Number two, we must establish a national system to provide for safe disposal of PCB's already used in electrical equipment. We were shocked to hear of the hundreds of millions of capacitors that are in use and we can just envision that over the next 30 to 50 years these elements are going to be disposed of without any control whatsoever, and eventually they are going to leak into our environment.

Number three, the Toxic Substances Control Act must be passed. We must avoid future problems of this type with other chemicals. I admit our industry has not worked hard enough to get this bill passed. We did support it as it went through the various stages of the Congress, but we did not really get behind it and scream loud enough. I can assure you we will do this from this point on.

Number four, we believe the present Food and Drug tolerance of 5 ppm in fish should be retained pending further research on the toxic effects on humans of PCB's. We believe that any reduction in tolerance should be selective, based on the role and the specific effect of the food in the diet.

Five, compensation, in all justice, must be made to commercial fishermen whose livelihood is destroyed by prohibition of sale of the species of fish in which the presence of PCB exceeds the Food and Drug tolerance. One element of our society must not pay for the mistake of the entire society.

And sixth, Congress should investigate the Environmental Protection Agency to determine why effluent standards for PCB's have not yet been established despite the clear mandate of the Water Quality Act to do so. If EPA doesn't have the funding, we have to get it. If the deadlines were unreasonable as set by Congress in 1972, then Congress should know they were unreasonable. Nonetheless, our industry would have expected results from this act at this time. Thank you.

PCB BODY BURDENS DENY FULL USE OF THE GREAT LAKES FISHERY RESOURCE

Carlos M. Fetterolf, Jr.*

The 1954 Convention on Great Lakes Fisheries between the United States of America and Canada led to establishment of the international Great Lakes Fishery Commission in 1956. The Commission has two major thrusts, control of the sea lamprey and determination of research and management strategies to provide sustained productivity in the convention area of any stock of fish which is of common concern to the parties. The Commission works cooperatively with the Canadian and U.S. Federal agencies and the provincial and State governments of Ontario and eight Great Lakes States to improve and perpetuate Great Lakes fishery resources.

The statement below is not Commission-approved. It is an expression of my personal opinions as the Commission's executive secretary.

The U.S. Food and Drug Administration guideline of 5 μg/g (ppm) in edible tissue of fish has been exceeded in numerous species in lakes Michigan, Huron, Erie, and Ontario, and in their connecting waters. Several important sport and commercial species are included with those that exceed the guideline. This situation casts a pall over the social and economic aspects of Great Lakes fisheries. It creates a very real problem for commerical fishermen, processors, and retailers; a shadow of doubt in the minds of every consumer and sport fisherman; an added question for the fishery manager; a symbol of defeat for the water pollution control agencies; and a mark for every environmental management critic to flaunt as an example of the failure of the "system." It denies full use of the Great Lakes fishery resource.

I hope none of you have the feeling that if residues in Great Lakes fish diminish to below 5 μg/g that all is well. A few years ago commercial mink ranchers fed their animals Great Lakes fish until they noticed a disturbing phenomenon, reproduction was falling off alarmingly. This led to studies of the effects of PCB's in ranch mink. Ringer et al. (ref. 1) demonstrated that 2 μg/g PCB's in fish flesh prevented survival of newborn animals, and Platonow and Karstad (ref. 2) demonstrated that reproduction was eliminated in mink fed a beef diet containing 0.64 μg/g Aroclor 1254, a PCB compound. If regulatory agencies consider fish as animal feed a use to be protected, application of a modest 0.2 safety factor provides a tissue level of 0.1 μg/g PCB in whole fish. There aren't many adult fish in the Great Lakes system that can meet that objective at this time.

The presence of PCB's in Great Lakes fish continues to deny full use of the fishery resource. The Great Lakes Fishery Commission is not a regulatory agency. We must depend on legislative action to pass the laws and enforcement agencies to furnish the muscle which will provide an aquatic environment that will produce usable fishery products. It appears to me that some foot dragging has been going on. How do we get response from a regulatory agency? Dr. Nisbet expressed disappointment that so few people were aware of his $8 publication and concluded that to have impact one must release large amounts of reprints into the environment. I don't think that's the answer.

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*Executive Secretary, Great Lakes Fishery Commission, Ann Arbor, Michigan.

How about development of data and publication by the regulatory agency itself? You heard Charlie Walker state that PCB's in fish were added in 1970 to the National Pesticide Residue Monitoring Program participated in by EPA. Data showing PCB body burdens as high as 213 µg/g in the Hudson River and 133 µg/g in the Ohio River were available 5 years ago. FDA guidelines of 5 µg/g were the same then as they are now.

How about the regulatory agency funding a study to recommend allowable levels of contaminants? EPA funded such a study by the National Academy of Sciences/Engineering in 1971 and the first draft was delivered on schedule to EPA's Washington office in December 1971. The recommendation for PCB's in that draft of Water Quality Criteria 1972 (ref. 3) read similarly to that in the published document:

> "Aquatic life should be protected where the maximum concentration of total PCB in unfiltered water does not exceed 0.002 µg/l at any time or place, and the residues in the general body tissues of any aquatic organism do not exceed 0.5 µg/g."

The National Academy report concluded that water quality levels alone were insufficient to predict what body burdens could result and therefore combined a body burden recommendation. If the body burden exceeded the recommendation, then obviously the water concentration was too high and should be further restricted.

In the four years since EPA received that recommendation from the National Academy of Sciences, sufficient data have been developed so that the recommendation of the Water Quality Objectives Subcommittee to the International Joint Commission made in June 1975 (ref. 4) could read:

> The concentration of total polychlorinated biphenyls in fish tissues (whole fish, calculated on a wet weight basis), should not exceed 0.1 micrograms per gram for the protection of fish consuming birds and animals.
>
> NOTE: The Subcommittee expresses concern that a water concentration objective for this ubiquitous contaminant is unavailable. Based upon poorly defined bioconcentration factors it may be concluded that PCB's in water should not exceed 0.001 micrograms per litre (1 ppt). However, this level may not be adequate to provide protection to certain predators, and could presently not be enforced because of insufficiently sensitive quantification limits.

This was a reduction to 0.1 µg/g body burden in whole fish from the NAS recommendation of 0.5 µg/g and a reduction in water level concentration from the 0.002 µg/l of the Blue Book to stating that 0.001 µg/l may be inadequate.

I am pleased that EPA is currently releasing for review their proposed Quality Criteria for Water. Remember yesterday Tom Kopp showed us the recommendation for PCB's, "0.001 µg/l for freshwater and marine aquatic life and for consumers thereof." Wastes go into systems, different systems have different capacities of response to PCB's. Is a blanket nationwide water level fair to industry, the environment, and the people? Will this water concentration alone do the job?

Apparently there is no accompanying body burden recommendation. This is disturbing to me. I didn't know EPA was so confident that bioaccumulation from 0.001 µg/l won't be a problem. Remember I told you that 0.64 µg/g Aroclor 1254 in the diet of ranch mink eliminated reproduction? A modest safety factor of 0.2 provided a recommended residue in fish of 0.1 µg/g used to feed mink. You heard several speakers mention bioconcentration factors greater than 100,000, some as high as 270,000. Two hundred thousand multiplied by a water concentration of 0.001 µg/l yields a body burden of 0.2 µg/g, double the recommended residue if you're in the business of selling fish to mink ranchers.

I don't believe the concentration of PCB in the waters of Lake Superior is known accurately enough that it appears in the refereed literature. It is generally believed to be 0.001 µg/l, the concentration recommended in EPA's proposed Quality Criteria for Water. An analytical chemist of EPA's National Water Quality Laboratory at Duluth on the shores of Lake Superior estimates the PCB concentration in Lake Superior water at 0.0004 µg/l, 0.4 parts per trillion. The total PCB body burden of whole Lake Superior adult ciscoes is 5 µg/g and greater. Depending on which water concentration one chooses, we have a bioconcentration factor of at least 500,000 times. I don't believe the proposed EPA water concentration is going to do the job necessary so that Great Lakes fishery resources can be fully used. Canada shares the Great Lakes with us. The November 17, 1975, announcement by its Department of Health and Welfare, lowering its PCB regulatory level to 2 µg/g in edible tissue is going to further restrict the full use of Great Lakes fishery resources.

You heard Dr. Munson say this morning that 99 percent of the PCB content in upper Chesapeake Bay water is associated with suspended sediment. Obviously it's going to affect analytical results if you measure water concentrations unfiltered, settled, or filtered. I was surprised EPA's proposed recommendation for PCB did not include a mention of whether suspended materials should be included in the analysis. By the way, are PCB's adsorbed to suspended materials biologically important? If not, perhaps the water should be filtered before analysis. Do regulatory agencies know the answers to questions so critical to our environment and economic welfare?

There are several avenues of problem solution open to EPA. There are tough choices, and fortunately it is not my responsibility to make a recommendation. All I ask is that something be done, and that what is done will permit full use of the Great Lakes fishery resource.

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4. Great Lakes Water Quality Board, third annual report to the International Joint Commission, 1975, IJC Regional Office, Windsor, Canada.

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DR. WILBUR P. MCNULTY (Oregon Regional Primate Research Center, Beaverton, Oregon): I will summarize briefly a series of experiments on the toxicity of polychlorinated biphenyls (PCB's) in rhesus monkeys. Three preliminary conclusions can be drawn from the results.

First, PCB's produce a unique and reproducible constellation of pathologic changes in monkeys over a wide range of doses. These changes are different from those reported for other laboratory animals. Furthermore, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) causes exactly the same constellation.

Second, rhesus monkeys are lethally poisoned by very low levels of intake of PCB's in the diet, roughly 100 times less than the levels which cause serious illness in rats.

Third, some individual PCB components are quite toxic for monkeys and some are not. Contaminating chlorodibenzodioxins or -furans probably cannot account for the toxicity of commercial Aroclor 1242 in monkeys.

Figure 1 shows extensive downgrowth of mucous glands into the submucosa of the stomach of a monkey which died after 8 months consumption of regular monkey chow to which Aroclor 1242 was added at 3 ppm. The development of this gastric lesion was studied by monthly biopsies in monkeys fed Aroclor 1242 at 3 to 10 ppm, and the results will be discussed by my colleague Dr. Bell.

An early clinical sign of PCB poisoning in rhesus monkeys is thickening and reddening of the eyelids. The Meibomian (sebaceous) glands were completely converted to squamous cysts (figure 2). Sebaceous glands associated with hair follicles in the face and and scalp similarly underwent squamous metaplasia, with atrophy of the gland or, occasionally, cyst formation (figure 3).

The thymus became markedly atrophic (figure 4); the thymocytic cortex disappeared entirely, and the corpuscles formed small cysts.

Exactly the same pattern of changes was found in monkeys accidentally poisoned with what subsequent chromatographic analysis of the tissues showed was probably Aroclor 1260, presumably used in construction materials in the pens. And finally, the same pattern followed experimental poisoning with TCDD—in only 12 days at an intake of 20 ppb in the diet.

The spectrum was the same at exposure of from 3 to 800 ppm of Aroclor 1242, though of course

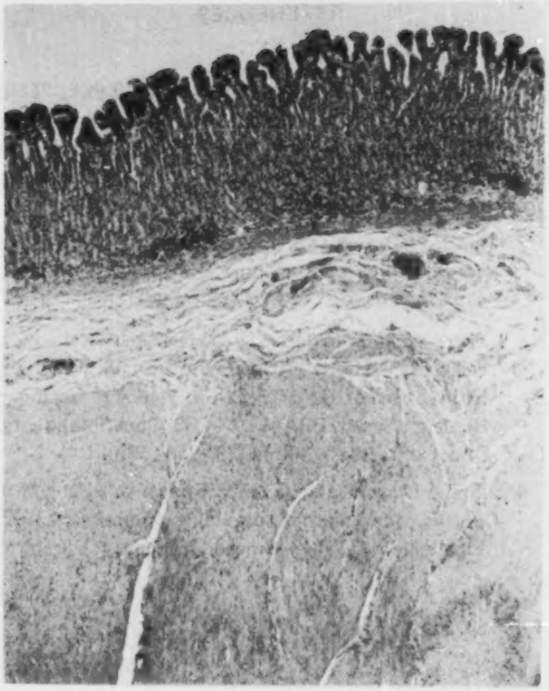

Figure 1a. Normal gastric mucosa of young male rhesus monkey. H&E, 30x.

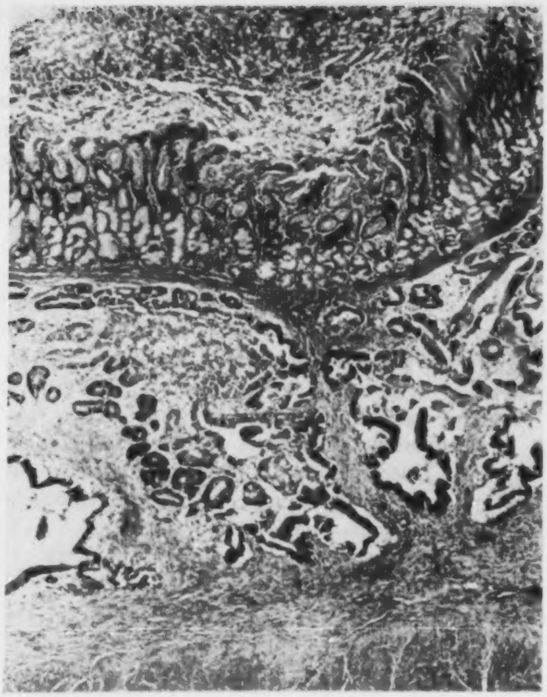

Figure 1b. Gastric mucosa of young male rhesus monkey fed diet containing 3 ppm Aroclor 1242 for 8 months. Extensive mucous epithelial invasion of submucosa. H&E, 30x.

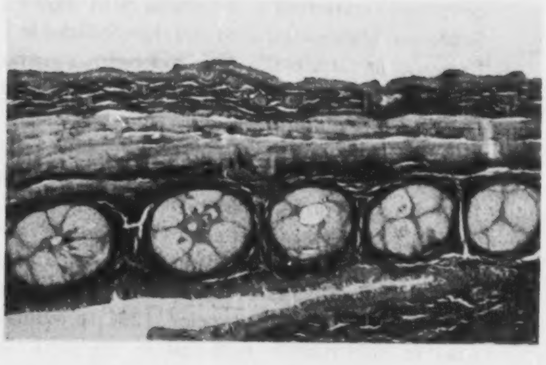

Figure 2a. Normal eyelid of young male rhesus monkey. Transverse section, conjunctival surface below. H&E, 30x.

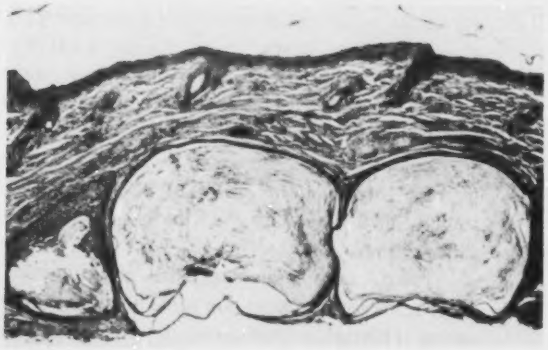

Figure 2b. Eyelid of young male rhesus monkey fed diet containing 3 ppm Aroclor 1242 for 8 months. Squamous metaplasia and cystic dilation of Meibomian glands. H&E, 30x.

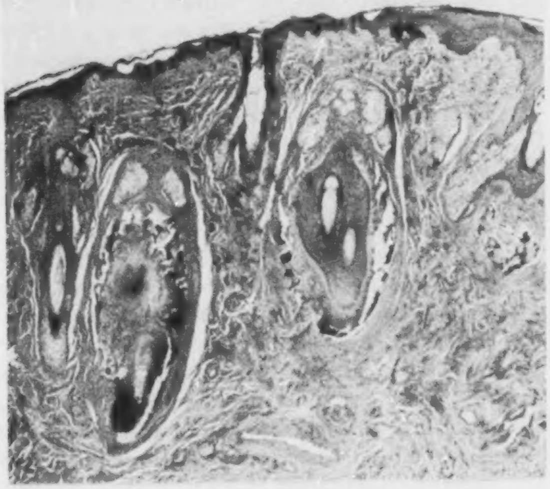

Figure 3a. Normal vibrissae or sinus hairs in lip of young male rhesus monkey. A garland of sebaceous glands encircles the hair follicle at the upper end of the blood sinus. H&E, 30x.

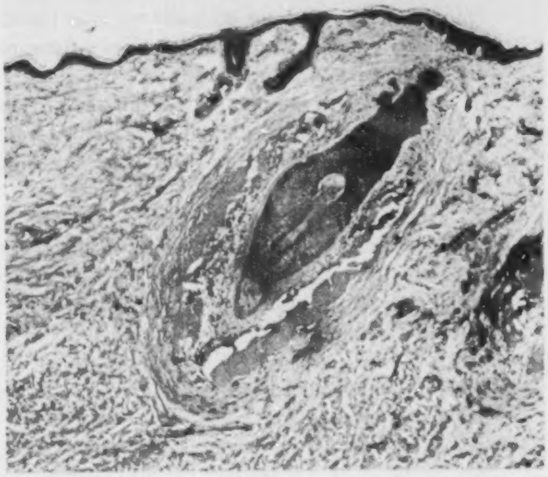

Figure 3b. Sinus hair of lip of young male rhesus monkey fed diet containing 3 ppm Aroclor 1242 for 8 months. Garland of sebaceous glands is absent. H&E, 30x.

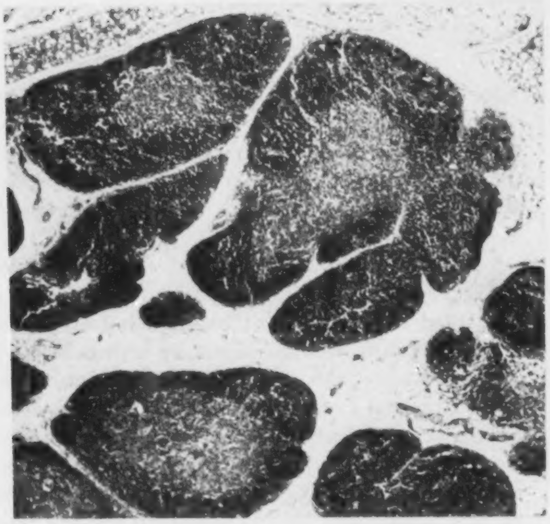

Figure 4a. Normal thymus of young male rhesus monkey. H&E, 30x.

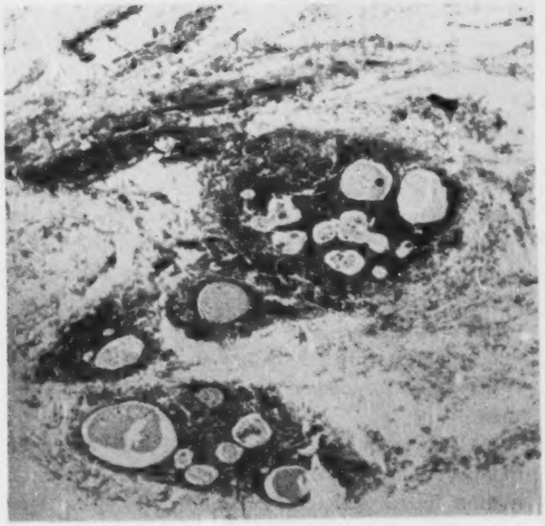

Figure 4b. Thymus of young male rhesus monkey fed diet containing 3 ppm Aroclor 1242 for 8 months. Severe atrophy and cyst formation in corpuscles. H&E, 30x.

illness and death came sooner at higher doses. A dietary level of 3 ppm corresponds to an intake of 150 μg/kg/day, or about 40 times the level that Dr. Nisbet calculated might be the intake of a fisherman or a nursing baby today.

At least with respect to the effects on sebaceous glands, the disease in monkeys resembles that reported in the victims of Yusho, an accidental poisoning of Japanese people.

I have tested three pure PCB compounds at 10 ppm; this is a reference level which, in the case of Aroclor 1242, causes barely discernible histologic changes in 30 days, slight clinical illness in 45 days, and outspoken disease in 60 days.

At this level, 2,4,4′-trichlorobiphenyl and 2,4,5,2′,5′-pentachlorobiphenyl, both major components of Aroclor 1242, caused no clinical or histological changes in rhesus monkeys experimentally fed for 80 days. On the other hand, 3,4,3′,4′-tetrachlorobiphenyl, which is not significantly present in Aroclor 1242 killed a monkey and caused the usual pathologic changes in 34 days. This finding invites the speculation that the metabolites of some PCB's may be the actual toxins. For example, 2,3,7,8-tetrachlorodibenzofuran (TCDF), which is known to be quite toxic for laboratory rodents but has yet to be tested in monkeys, could conceivably be formed from the 3,4,3′,4′-tetrachloro-biphenyl by hydroxylation and condensation. But this lot of tetrachlorobiphenyl has yet to be analyzed for possible preexisting contaminants.

However, it is not likely that contaminating dioxins or furans can account for the toxicity of commercial PCB's. Pilot experiments have indicated that TCDD is about 10,000 times as toxic as Aroclor 1242 on a per gram basis. TCDF can be expected to be somewhat less active. Since our analysis of Aroclor 1242 has shown contamination to be not more than 1 ppm, there is not enough dioxin or furan present to account for the effects of the Aroclor.

ULTRASTRUCTURAL FEATURES OF GASTRIC MUCOSA AND SEBACEOUS GLANDS AFTER INGESTION OF AROCLOR 1242 BY RHESUS MONKEYS

Mary Bell, Ph.D.

The effects of PCB's on the stomach, skin, and liver of rhesus monkeys have previously been described by Allen and his associates (refs. 1-5) and some of these effects by McNulty (ref. 6). Of these organs, the liver has been the only one on which any electron microscopic observations have been made. In the liver, PCB administration results in a proliferation of the smooth endoplasmic reticulum and, in some cases, the accumulation of fat droplets within the hepatocyte. Because the cytological features of these organs are extremely diverse, it is important to document the effects of PCB's of all of them.

McNulty (ref. 6) has described a series of rhesus monkeys that were fed either 3, 10, 30, or 100 parts per million (ppm) Aroclor 1242 until they became moribund. The stomach and lip of each animal were serially biopsied at monthly intervals beginning 2 weeks after first ingestion of these compounds. The tissues were processed according to routine procedures for light and electron microscopic observation. This report describes the effects of ingestion of Aroclor 1242 on the cellular components of the stomach and of the sebaceous glands associated with the large tactile, facial hairs, vibrissae, of these animals.

Figures 1 and 2 demonstrate the changes seen with the light microscope in the gastric epithelium of an animal fed 100 ppm Aroclor 1242. After 2 weeks ingestion, the stomach was still essentially normal (figure 1). The mucus-secreting surface and the gastric glands that contain parietal cells (hydrochloric acid secretors) and zymogenic cells (enzyme secretors) showed no pathologic changes. After ingestion of 100 ppm PCB's for 2 months, however, the mucus-secreting surface had become hyperplastic (figure 2); the parietal and zymogenic cells of the gastric glands had totally disappeared and had been replaced by mucus-secreting cells. These effects, though demonstrable at varying times after onset of PCB administration, were identical in all animals at any of the dose levels used. Figures 3 and 4 show the

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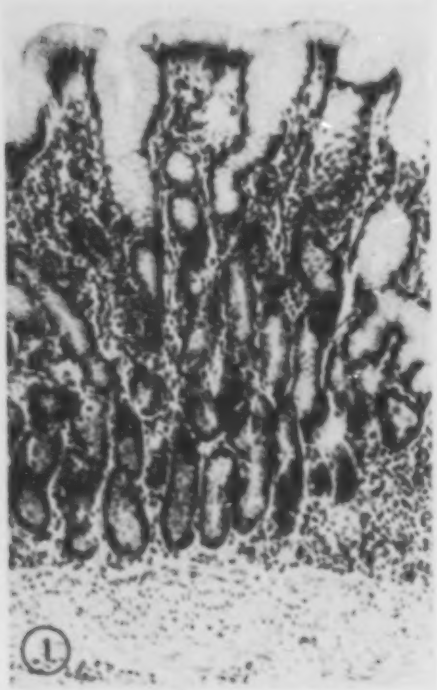

Figure 1. A light micrograph of the gastric mucosa of a rhesus monkey fed 100 ppm Aroclor 1242 for 2 weeks. The mucus-secreting surface mucosa and the gastric glands containing parietal and zymogenic cells are of essentially normal appearance (x140).

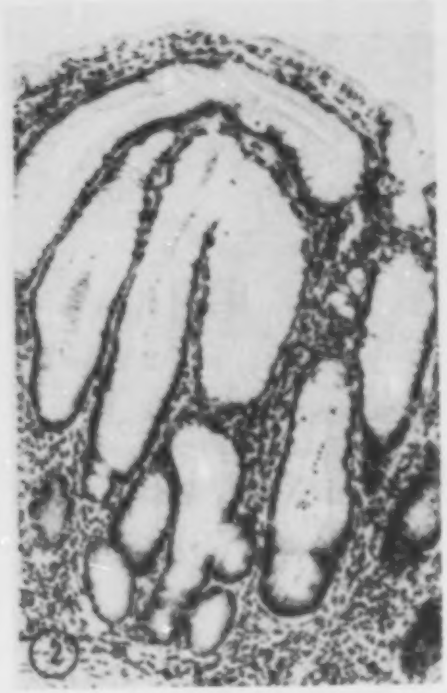

Figure 2. An oblique section of gastric mucosa from a rhesus monkey fed 100 ppm Aroclor 1242 for 2 ½ months. Most of the gastric glands have been replaced by mucus-secreting cells (x140).

effects after 5-½ months on the stomach of an animal fed 3 ppm Aroclor 1242. At this time, most of the gastric glands had been replaced by mucus-secreting epithelium.

At the electron microscope level, the first PCB-related changes were seen in the parietal cells of the gastric glands, and some of these changes were observed at the higher dosage levels shortly after the onset of PCB ingestion. These cells normally have a distinctive cytology, which includes intracellular canaliculi through which the hydrochloric acid is secreted, many small cytoplasmic vesicles, and large numbers of mitochondria. After PCB ingestion, the intracellular canaliculi became somewhat distended, the cytoplasmic vesicles became less discrete and tended to break down, and small dense bodies, presumably containing hydrolytic enzymes, accumulated (figure 5).

In the bases of the gastric glands, mucus-secreting cells were interspersed among zymogenic cells after ingestion of PCB's. Zymogenic cells normally contain extensive concentrations of granular endoplasmic reticulum located basally in the cells; this reticulum is responsible for the production of enzymes. After PCB ingestion, however, mucus-secreting cells with typically little endoplasmic reticulum began to appear adjacent to the zymogen-secreting cells (figure 6). In some cells still recognizable as zymogenic, the endoplasmic reticulum was dilated and the cells contained large autophagic vacuoles (figure 7). The latter usually occur in cells that are undergoing degradation.

Figures 8 and 9 show the changes seen at the light microscope level in the sebaceous glands associated with the tactile hairs of the face after PCB ingestion. Normally, lipid production by the sebaceous glands begins close to the peripheral portions of the glandular alveoli and continues to the more central portions where the

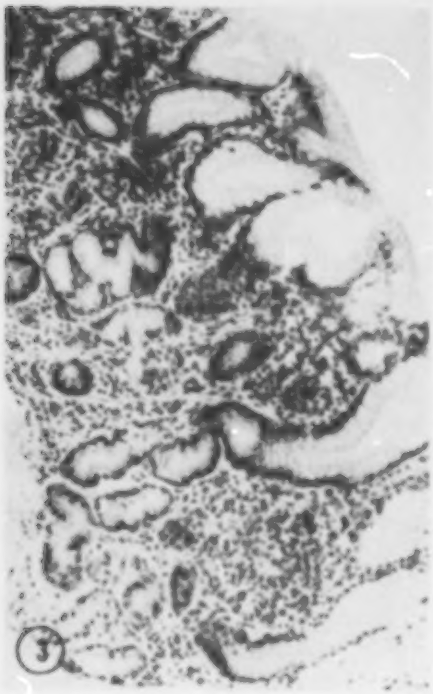

Figure 3. Gastric mucosa of a rhesus monkey fed 3 ppm Aroclor 1242 for 5 ½ months. Most of the gastric glands have been replaced by mucus-secreting cells. The effects are similar to those shown in figure 2 (x140).

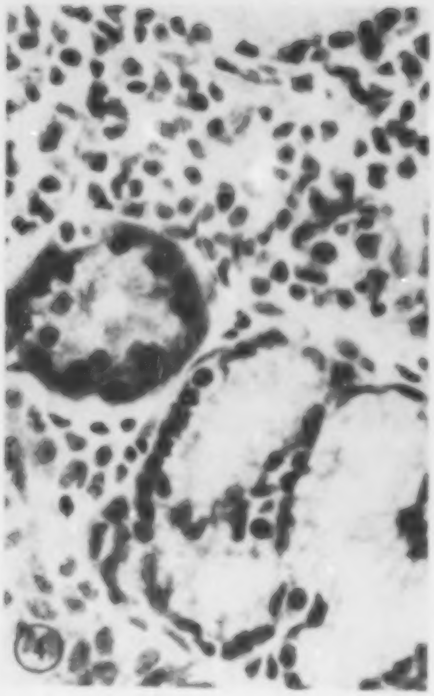

Figure 4. A higher magnification of a portion of figure 3. The gastric glands are almost completely composed of mucus-secreting cells. Only one gland (at the left center of the field) contains zymogenic cells (x560).

lipid is secreted onto the hairs. After ingestion of Aroclor 1242, lipid synthesis and accumulation became increasingly limited to the central zones of the alveoli; ultimately, only small buds of cells no longer secreting lipid droplets were found adjacent to the hair follicles in the sites normally occupied by sebaceous glands. These effects were identical in all animals fed PCB's, but they occurred most rapidly at 100 ppm, least rapidly at 3 ppm Aroclor 1242.

As observed at the electron microscope level, sebaceous cells normally contain large Golgi zones that are the site of synthesis of the lipid droplets and many profiles of agranular endoplasmic reticulum, which are considered to participate actively in the production of lipid (figure 10). After PCB ingestion, these membranes acquired a softened appearance during stages when they were still prominent. Many small electron-opaque bodies, probably indicative of the presence of hydrolytic enzymes, also occurred in these cells after PCB ingestion. When no lipid droplets were detectable after PCB ingestion (∿ 2-½ months at 100 ppm, ∿ 4-½ months at 3 ppm), filaments usually associated with epidermal-type cells (from which sebaceous glands are embryologically derived) often increased in abundance in the sebaceous cells, they continued to contain a few profiles of granular endoplasmic reticulum, but smooth membranes could no longer be detected (figure 11). Ultimately, the sebaceous cells no longer even resembled the epidermal cell type, and they no longer appeared capable of producing lipid.

These studies do not enable us to determine whether cells can change their direction of differentiation under the influence of PCB's or whether they merely differentiate in the only direction available to them when PCB's are present. They do tell us, however, that cells, depending on their location and function in the body, respond in very different ways to PCB's.

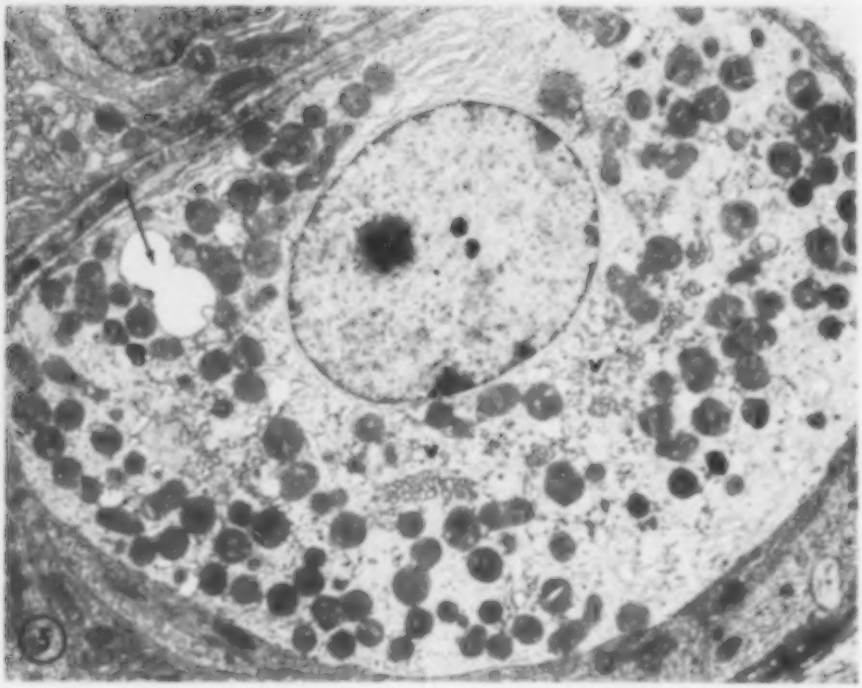

Figure 5. An electron micrograph of a parietal cell from the stomach of a rhesus monkey fed 30 ppm Aroclor 1242 for 2 weeks. Many small perinuclear cytoplasmic vesicles (v) are disrupted and a portion of an intracellular canaliculus is distended (arrow) (x6300).

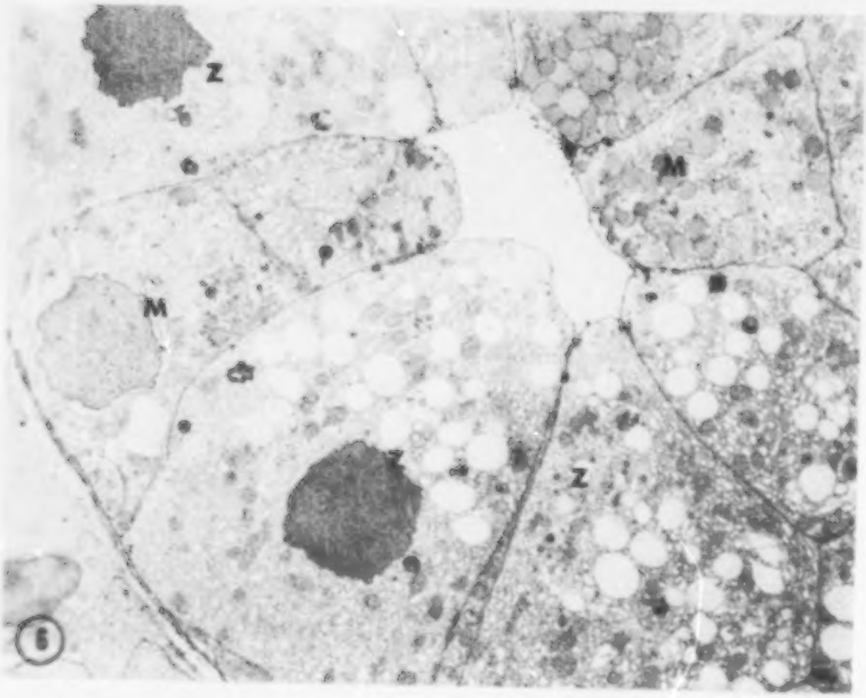

Figure 6. An electron micrograph of a portion of a gastric gland from a rhesus monkey fed 10 ppm Aroclor 1242 for 4 ½ months. Mucus-secreting cells (M) have become interspersed with zymogenic cells (Z) in this location (x3400).

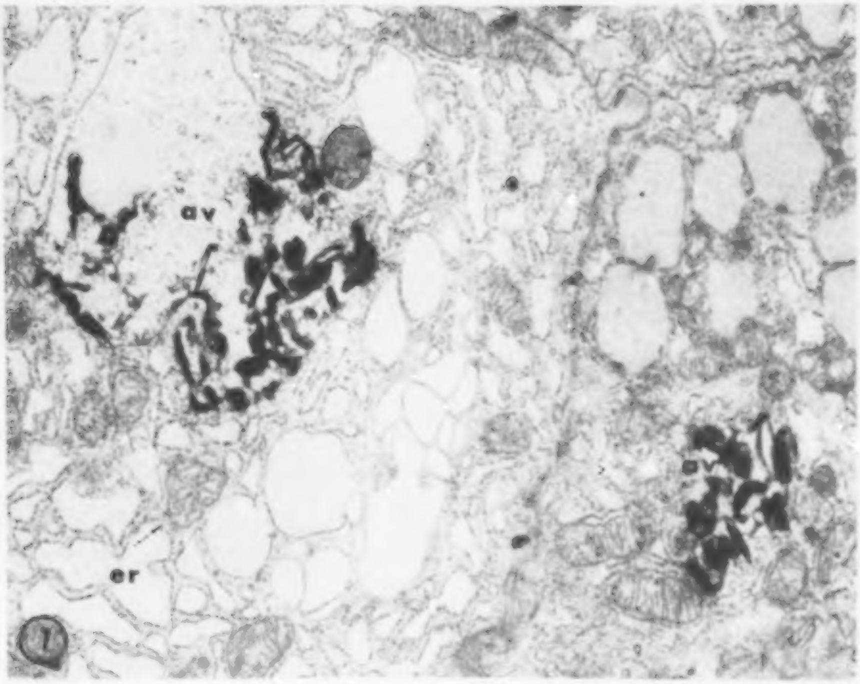

Figure 7. Portions of two zymogenic cells containing large autophagic vacuoles (av) and dilated granular endoplasmic reticulum (er). The animal from which the tissue was biopsied had been fed 3 ppm Aroclor 1242 for 5 ½ months (x13,150).

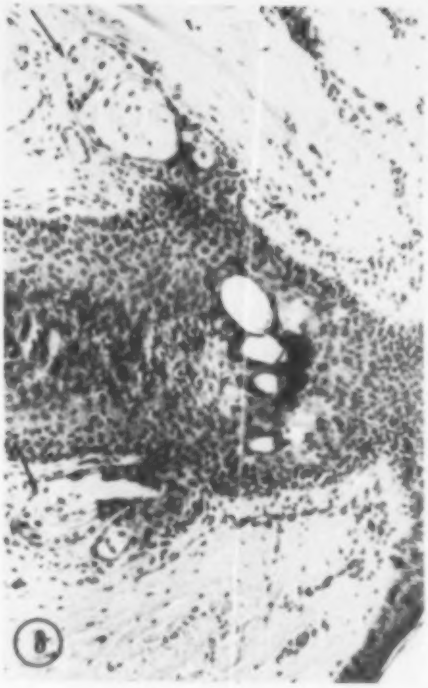

Figure 8. An oblique section through a tactile hair with associated sebaceous glands. Though the glands are small, cell differentiation, evidenced by sebum droplets, is still occurring close to the periphery of the alveoli (arrows). This tissue was taken from an animal fed 3 ppm Aroclor 1242 for 3 ½ months (x140).

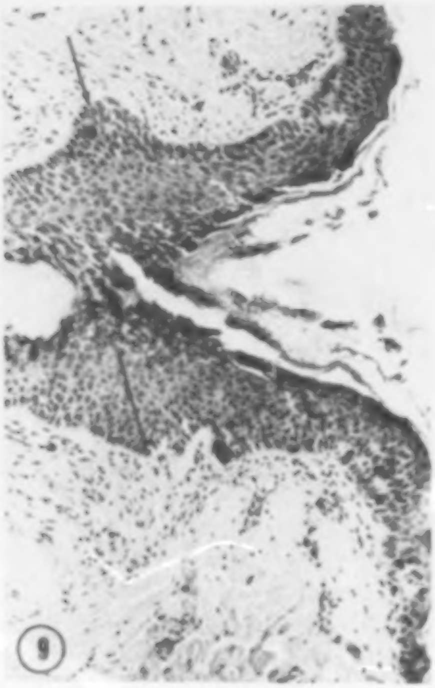

Figure 9. A light micrograph of a comparable site as that shown in figure 7 but after 3 ppm Aroclor 1242 for 4 ½ months. No sebum droplets or differentiating cells are visible in the sites (arrows) normally occupied by the sebaceous glands (x140).

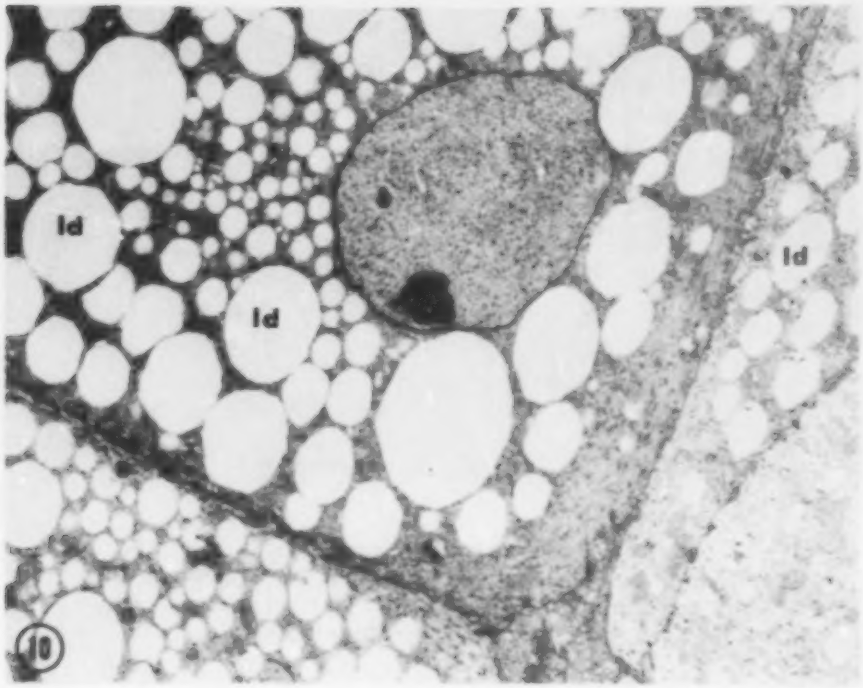

Figure 10. An electron micrograph of cells with essentially normal features in a sebaceous gland associated with a large tactile hair. The animal from which the tissue was biopsied had been fed 30 ppm Aroclor 1242 for 2 months. Although these cells are close to the periphery of an alveolus, they are producing abundant lipid droplets (ld) (x5600).

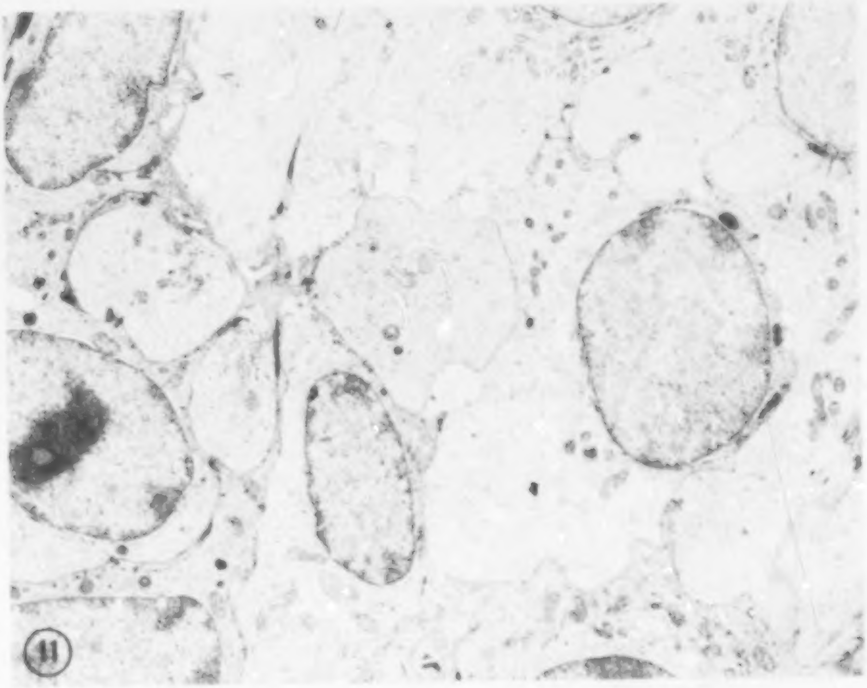

Figure 11. An electron micrograph of cells from a site similar to that shown in figure 10, but from an animal fed 3 ppm Aroclor 1242 for 4 ½ months. The cells contain no lipid droplets, and their cytoplasm appears largely amorphous (x7200).

ACKNOWLEDGMENT

All of the specimens and much of the data for this study were collected at the Oregon Regional Primate Research Center. Work was supported in part by Grants AM08445 and RR00163 of the National Institutes of Health and ES-00159-10 of the National Institute of Environmental Health Sciences.

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THE VIEW OF THE PAPER INDUSTRY ON THE OCCURRENCE OF PCB'S IN THE ENVIRONMENT AND THE NEED FOR REGULATION

Paul E. Trout*

My name is Paul E. Trout. I am Director of Environmental Control for Container Corporation of America and am presenting the following comments on behalf of my company and the American Paper Institute.

The American Paper Institute is a national organization composed of manufacturers of pulp paper, and paperboard. Members utilize both virgin and recycled fiber in the manufacture of paper products. In 1973, the American paper industry and its wastepaper suppliers recycled nearly 15 million tons of wastepaper (ref. 1) an all time high. In the same year, approximately 41 percent of the fiber used by Container Corporation of America's 13 paperboard mills was derived from wastepaper.

This recycling of wastepaper unwittingly plunged my company and similar recyclers into the morass of the PCB problem in late 1970.

I. THE PAPER INDUSTRY DOES NOT USE OR INTRODUCE PCB'S INTO THE ENVIRONMENT BUT RATHER RECIRCULATES PCB'S ALREADY PRESENT, WHICH ARE UNAVOIDABLY INCLUDED IN ITS MANUFACTURING INPUT

PCB's are not a part of the paper manufacturing process. Consequently the paper industry does not add new PCB's to the environment. PCB's are, however, unavoidably included in certain inputs into the paper manufacturing process. In particular, the water supply used in paper making contains PCB's from other unrelated sources. The paper manufacturing process is not designed to remove PCB's and consequently effluents do contain low levels of this chemical which have entered the mills in the intake waters.

The other PCB input to the paper manufacturing process is the raw material used by recycling mills as a fiber source. Until early 1971, NCR paper (carbonless copy paper) used Aroclor 1242 enclosed in microcapsules coated on the paper. Thus business forms made with carbonless copy paper have been a component of office waste paper, a portion of which is recycled in the papermaking process. Inevitably the PCB's present in the office waste have been introduced into the paper recycling process.

Again, no new PCB's are added to the environment by paper mills but, rather, existing PCB's are simply recirculated. If the paper mills could not recylce the office waste containing PCB-bearing paper, the paper would just as surely enter the environment either through leaching in landfills or volatilization in incinerators which are unable to destroy the PCB's. In either of those events, municipal solid waste systems would have a very substantial new burden added to their normal operating loads and the important national recycling would be significantly injured. Moreover, the PCB present in the old NCR paper still occasionally finding its way into the waste stream is Aroclor 1242, the one not accumulating in the environment. It is this PCB that is found both in the paper products and in the effluent of paper mills.

Since 1971, when the use of PCB in the manufacture of carbonless carbon paper was halted, PCB levels in recycled-fiber paperboard used for food packaging have continued to decline. The industry monitoring program showed that, in the third quarter of 1975, 99.63 percent of such paperboard had a PCB content less than 10 ppm and 91.3 percent had a PCB content less than 2.5 ppm. These results have been achieved through the most careful selection of the wastepaper used in the recycling process with rejection of all types of wastepaper suspected to contain PCB's.

II. THE PCB RECIRCULATED BY THE PAPER INDUSTRY, AROCLOR 1242, IS NOT FOUND TO ACCUMULATE IN ANIMALS, MAN, OR IN THE ENVIRONMENT

The PCB occasionally detected in paper products and in the effluent of paper manufacturing plants is Aroclor 1242. For the years 1960 through 1971, Aroclor 1242 represented from 48.15 to 68.9 percent of the total domestic PCB production, reaching this maximum percentage in 1967 and 1968 and decreasing thereafter. Accordingly, it would necessarily be the PCB most widespread in the environment if all PCB's were all equally degradeable. In fact, however, Aroclor 1242 is neither found in the tissues of man or animal nor in the aquatic environment generally. This indicates that the lower chlorinated PCB's such as Aroclor 1242 are either metabolized, decomposed in aqueous environments, or degraded by photolysis. If accumulation of PCB in aquatic environments, and thereby in the food chain and man, is perceived to be a potential threat to human health, it is a threat unrelated to Aroclor 1242. Any PCB regulation based on environmental needs must accomodate this fact.

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III. THE CURRENT LEVELS OF DIETARY INTAKE IS MANY ORDERS OF MAGNITUDE LOWER THAN THE ALLOWABLE DAILY INTAKE AS FOUND BY THE FOOD AND DRUG ADMINISTRATION

Evaluation of the need for regulatory controls of the source and distribution of PCB's must begin with the threat PCB's pose to the health of man or other parts of the living environment. In the case of PCB's, the possible threat to human health is through the food supply. Accordingly, that evaluation should start with the levels of dietary intake of PCB's with reference to the levels posing a threat to man.

As a distinguished committee of the National Academy of Sciences, National Research Council stated, "For every chemical there is some finite level sometimes called the 'safe' level, at or below which it can be present in food without prejudicing safety (ref. 2)." Conversely, as three other NAS committees have recently noted, "There is no substance which under certain circumstances, could not be dangerous and unsafe (ref. 3)." The possible need for regulatory controls of PCB's therefore must be assessed in terms not of the conceivable danger posed by some amount of a substance but rather in terms of the available margin of safety between conceivably harmful dosages and actual consumption.

According to the Food and Drug Administration's Director of the Office of Science, Dr. Albert C. Kolbye, Jr., the allowable daily intake of PCB's in the adult human diet is 200 μg/g/day. This conclusion is based upon Dr. Kolbye's analysis of available animal toxicity studies including the recent investigations of Dr. Kimbrough as well as upon analysis of human toxicity data arising from the "Yusho" incident in Japan involving massive intoxication by PCB's.

The Food and Drug Administration's "Total Diet Studies" demonstrate that PCB average dietary intake levels have never been remotely near this 200 μg/adult/day figure. On March 18, 1972, FDA stated in a *Federal Register* notice that its total diet studies indicated an average daily intake of 7/μg/day/adult or 4.7 ppb in the total diet. This is 3.5 percent of the amount Dr. Kolbye concluded was plainly safe. Those data reflected the period of August 1969 through March 1972 when PCB exposure must have been at its peak since the steps taken by Monsanto Company in early 1971 to confine the sale of PCB's to closed systems had not yet had time to be reflected in the data base.

Fifteen months later, on July 6, 1973, FDA announced that the quantitatively measurable residue of PCB's were equivalent to an average dietary intake of 4.2 μg/day/adult. This corresponds to a dietary intake of 2.8 ppb or about 2 percent of the level Dr. Kolbye identified as safe. According to FDA's total diet studies for fiscal year 1973, the dietary intake of PCB's was 1 μg/day/-adult or .67 ppb. This is less than ½ percent of the safe amount calculated by Dr. Kolbye. According to these studies for the period of October 1972 to January 1975, the average dietary intake was .178 μg/day/adult or .118 ppb (118 ppt), or below 1/10 of 1 percent of Kolbye's safe level. An examination of the total dietary intake as shown by FDA's total diet studies from January 1, 1974, through June 1975 shows PCB intake has been *zero*: that is, not a single quantifiable residue of PCB has been detected in the total diet study which would allow any quantitative estimate whatsoever.*

In short, dietary intake of PCB has been declining rapidly since approximately the time Monsanto implemented its voluntary controls on the distribution of PCB's to its customers. Even during the maximum exposure period, the average dietary intake was nowhere near what the Food and Drug Administration states to be an allowable daily intake. At the present time, there is an even vaster gulf—many orders of magnitude—between the allowable daily intake and the amount of PCB posing any conceivable hazard to man.

IV. ANY ADDITIONAL CONTROLS MUST ACHIEVE MAXIMUM ENVIRONMENTAL COST-EFFECTIVENESS

The paper recycling industry has been a participant in the PCB drama through circumstance, not by choice, and our industry is not alone in this. We thus appreciate the need for procedures to prevent similar happenings in the future as well as to correct remaining PCB problems.

Our industry approves the principle of restricting the use of hazardous substances. We do not, however, support far-reaching regulation of the nature of Senate Bill S776. Such legislation simply would amount to the "Biological and Analytical Chemist's Relief Act of 1975" and would, in addition, open a new Pandora's box of legal mischief. We believe duplication of existing legislation should be avoided and that deficiencies in existing legislation be corrected by specific amendment in order to maximize environmental benefit at minimum cost to the public.

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*It is also significant that for FY 1975 the total diet studies did not detect a single trace of Aroclor 1242, the PCB associated with the recycled paper manufacturing process.

We particularly urge States now considering new regulations to combat the PCB problem to develop rational rather than simplistic solutions. Destruction of the wastepaper recycling industry will benefit neither the public health nor the public purse.

We commend the Environmental Protection Agency for establishing a task group to investigate PCB imports. We urge that they continue to track down specific uses of these materials in the United States and publish this information so that further unknowing use of PCB's can be avoided.

We urge EPA to publicize abroad the self-regulatory action taken by Monsanto Co. to minimize further PCB contamination of the environment. Hopefully, such enlightened action may spread worldwide.

And, finally, we urge the Environmental Protection Agency to implement a program whereby PCB-containing wastepapers issuing from Federal agencies are disposed of by incineration at a temperature sufficient to destroy PCB. If Aroclor 1242 is an environmental hazard, this procedure would prevent the reservoir of this material now residing in the files of the world's greatest recordkeeper from contaminating the environment.

All of these control actions can be initiated immediately, without need for additional legislation and with immediate positive results.

To conclude, I would like to summarize these points:

I. The paper industry does not use or introduce PCB's into the environment but rather recirculates PCB's already present which are unavoidably included in its manufacturing input.

II. The PCB recirculated by the paper industry, Aroclor 1242, is not found to accumulate in animals, man, or in the environment.

III. The current levels of dietary intake is many orders of magnitude lower than the allowable daily intake as found by the Food and Drug Administration.

IV. Any additional controls must achieve maximum environmental cost-effectiveness.

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1. F. L. Smith, Jr., "Wastepaper Recycling: Review of Recent Market Demand and Supply," *Pulp and Paper*, September 1975, p. 148.
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3. "Principles for Evaluating Chemicals in the Environment," a joint report of three NAS committees: Committee for the Working Conference on Principles of Protocols for Evaluating Chemicals in the Environment; Environmental Study Board, NAS, National Academy of Engineering; and Committee on Toxicology, National Research Council, Washington, D.C., p. 83.

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MR. BERNARD A. KERNS (Westinghouse Electric Corporation, Pittsburgh, Pennsylvania): I very much appreciate the opportunity to make these comments at this conference. It is the hope of Westinghouse that this conference will bring together all of the scientific and technical information that is available to identify the positive aspects of the current mixtures that are being used by the electrical industry.

The Westinghouse Transformer Divisions and the Westinghouse Distribution Apparatus Division are users of a substantial quantity of polychlorinated biphenyls in the production of transformers and capacitors. At this point I should point out that Westinghouse uses a term inerteen for the mixtures of PCB's it uses as a dielectric, and that the generic term used in the electrical industry is askarel. These terms are really not interchangeable with the term PCB's. While there are 209 isomers of PCB's; the electrical industry uses only several mixtures of PCB isomers as dielectric.

Of these, Westinghouse utilizes primarily only two askarels, Aroclor 1242 for transformers and Aroclor 1016 for capacitors as inerteen. Since the early 1970's, when scientific studies indicated that PCB mixtures presented some hazard in the environment, Westinghouse has expended considerable money and effort to reduce the amount that might escape into the environment.

Measures that have been employed are: The sealing of drains in manufacturing areas where inerteen is used; utilizing specially designed incineration facilities for the destruction of scrap inerteen and special scientific landfills for the disposal of inerteen-contaminated materials; instructing operation personnel and our customers regarding the need for care and that special waste disposal is required; and reducing the number of pounds of inerteen per KVA in transformers and capacitors. Most of these measures were implemented prior to the enactment of the Federal Water Pollution Control Act amendments of 1972. It is significant to note that France has just enacted regulations on the use of PCB's which require actions that have been taken by Westinghouse since 1972.

Westinghouse recognized that these measures could not prevent the total elimination of inerteen escaping into the environment. Therefore, concurrent with the above measures, we have conducted extensive evaluation programs designed to utilize those mixtures of PCB's having low persistence and high biodegradibility in the environment.

By February 1968, Westinghouse determined that a mixture of PCB's sold by Monsanto as Aroclor 1242 would be satisfactory as inerteen for transformers. Aroclor 1242 contains about 91 percent of the lower isomers, containing four chlorines or less that more readily biodegrade in the environment.

This material has been used by Westinghouse since that time, with the understanding that over 90 percent of the small amount that did enter into the environment would have relatively low persistence. Monsanto subsequently developed a new material from Aroclor 1242 which contains more of the lower chlorinated isomers and marketed this material as Aroclor 1016. By the first quarter of 1972, this material was introduced by Westinghouse into the manufacture of all capacitors.

This material contains 99 percent of the lower biodegradible isomers, four chlorines or less, so that less than 1 percent of this material that might escape into the environment might be more resistant to biodegradation. The industry, Monsanto, our own research, scientific literature, and most of the papers presented at this conference have indicated that the lower chlorinated mixtures of polychlorinated biphenyls are more biodegradible and do not present the same long-term toxic environmental problems as those which contain the higher chlorinated isomers.

It is interesting to note, in this regard, that EPA researchers have found a significant difference between the effects of Aroclor 1242 and Aroclor 1016 on rats, confirming our position. But they published this information in Great Britain and did not make the information available during the toxic hearings of 1974. We believe the U.S. scientific community should determine the benefits and the environmental impact of the two or three askarels used by the electrical industry on a scientific basis and not let the desire to see a toxic substance bill passed stampede us into a selection of an alternate fluid which is more potentially dangerous to man and to his environment. Thank you.

STATEMENT RELATING TO POLYCHLORINATED BIPHENYLS ON BEHALF OF THE WISCONSIN PAPER COUNCIL

James S. Haney*

The Paper and Paper Recycling Industries in Wisconsin

I appear today on behalf of the Wisconsin Paper Council, the trade association for the pulp and paper industry within Wisconsin. I am chairman of the Wisconsin Paper Council's Government Relations Committee.

There are 49 pulp and paper mills in Wisconsin employing 46,000 people who produce in excess of 5 million tons of pulp and paper products annually. Wisconsin manufactures more than 11 percent of the total production of pulp and paper products in the United States and is the number one paper-producing State in the country.

Not only do we lead the nation in *papermaking*, but we are also the number one State in paper *recycling*. At least 19 Wisconsin paper firms recycle fibers to some extent and several firms make a specialty of it. A *fifth* of Wisconsin's annual paper production, about 780,000 tons, is made from *recycled, post-consumer wastes*. Some Wisconsin papermakers, like my own company, have been producing recycled paper and paperboard since the turn of the century.

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*Public Affairs Director, Bergstrom Paper Company, Neenah, Wisconsin.

What Has The Wisconsin Paper Industry Done For The Environment?

Manufacturing any product is not a clean, tidy process. Generally, it is noisy, usually it is dirty, and often it involves work with large machinery. It normally takes skilled labor, of which Wisconsin is fortunate to have an ample and sufficient supply. The point being, however, that in the manufacturing process certain natural resources are consumed and related byproducts are discharged or emitted as a part of the process itself.

Recognizing this impact on the environment upon which our industry depends, the Wisconsin paper industry had expended approximately $126 million on water pollution abatement equipment prior to 1975. It is estimated by the industry that within the next 5 years an additional $153 million, at a minimum, will be expended for a total investment of approximately $280 million for water pollution abatement equipment alone, not one cent of which is attributable to new production equipment.

As to air pollution, prior to 1975, the Wisconsin paper industry had invested approximately $34 million in air pollution abatement equipment and anticipates that between 1975 and 1980 it will invest minimally an additional sum in excess of $60 million. In rough figures, therefore, the paper industry in Wisconsin alone will have spent approximately $375 million by 1980 for air and water pollution abatement equipment—approximately $8,200 per employee—on what is very environmentally necessary, but absolutely unproductive, equipment.

The Issue of Polychlorinated Biphenyls

In light of previous comments at this conference, I think I need spend little time in identifying polychlorinated biphenyls (PCB's) or their historical uses. One of the many varied uses of PCB's prior to 1971 had been in carbonless copy paper, which has accumulated in the files of many businesses and numerous governmental agencies. From time to time, some of this paper is discarded and becomes part of the solid waste chain.

At this time I wish to make one point very clear. *Wastepaper collectors and recyclers do not manufacture PCB's nor do they use them in their manufacturing process*. To the extent that recyclable wastepaper does contain traces of PCB's, this contaminant is introduced into the industrial system and becomes part of the industrial waste discharge of recycling facilities. The only other known PCB's on a paper mill's premises might be PCB-containing transformers or capacitors.

The Wisconsin Paper Council is vitally concerned that overly restrictive regulations of PCB's, both in finished paper or paperboard products or in effluent discharges, could destroy the Nation's recycling efforts. We strongly urge that those who are drafting regulations concerning PCB's more fully analyze the impact of such regulations on recycling industries.

You must understand, for example, that a very small portion of the total amount of PCB's manufactured domestically by Monsanto Company were used for carbonless paper, which is the chief assumed source by which wastepaper contains the PCB contaminant. Aroclor 1242, by weight containing relatively lower amounts of chlorine than other domestically produced and sold PCB's, was used in the production of carbonless paper. Between 1958 and 1971, at which time the use of PCB's in the manufacture of carbonless paper was ceased, approximately 48 percent of Monsanto's PCB production was of Aroclor 1242. However, of its entire domestically sold production during those years, only about 22 percent was used for "plasticizer applications." Plasticizer applications include not only use for carbonless paper, but also for adhesives, textile and other surface coatings, inks, investment casting wax, and sealants. Therefore, we know that something considerably less than 10 percent of Monsanto's total PCB production between 1958 and 1971 was used in the manufacture of carbonless paper, all of it being of the Aroclor 1242 variety.

Aroclor 1242 has significantly different structural constituent characteristics, whereby its stability and persistence are less and its degradeability appears to be significantly higher than its comparatively more highly chlorinated Aroclor relatives. At least 1,500 reports in scientific literature have been written relating to PCB's and, whereas most of these deal with only the question of reporting the presence of PCB's in the environment, invariably these reports describe and relate to Aroclor 1254 and 1260, the more highly chlorinated PCB's produced by Monsanto. In fact, a Monsanto spokesman has indicated that it was his opinion that "if we could just turn the clock back" and have produced only Aroclors 1221, 1232, 1242, and no others, we probably wouldn't be holding hearings today. We are told that the principal Aroclors being found in Wisconsin fish are Aroclor 1254 (Mississippi River) and Aroclor 1248 (lower Green Bay). Predominantly it is Aroclor 1254 that is found in the environment. In terms of degradation and degradeability, there is a definite difference between Aroclors 1242 and 1254.

On the issue of bioconcentration, the EPA has stated that Aroclor 1242 bioaccumulates on the order of 8 times less than the more highly chlorinated Aroclors. On the issue of removal from effluent, the EPA has predicted that we can look forward to significant reductions in discharge concentrations as the program and projected treatment systems required by the NPDES program become effectively operational.

All of the above can be restated concisely as follows. Only a small portion of total PCB's domestically produced were utilized for purposes of manufacturing carbonless paper and, as to those particular Aroclors, they are relatively less stable and persistent, appear to be more degradeable, and are less susceptible to accumulation in fish than are their related, more highly chlorinated Aroclors. *As a result, the impact of the waste paper recycling industry on the issue of PCB's must be put into a more proper and positive perspective.*

Concluding Remarks

I think the time has come for us to recognize that we all know less than we would like to know about PCB's. Because of their persistence and because of uncertainties about their impact, the new production of this substance has all but been eliminated and is now used for only very specific purposes. *An inventory must be made of where PCB's are coming from; the type of Aroclors that are being discharged, emitted, and landfilled; the best methods of decreasing these discharges, emissions, and landfilling; and to put some proper perspective on the existence of this substance in our environment.* We know of no current practical method by which to separate out of the wastepaper mass that portion of paper that contains PCB's—particularly since PCB's have been recycled into paper products other than carbonless paper. *PCB's cannot be legislated out of the recycled paper system.* The whole concept of recycling wastepaper is at stake in considering the promulgation of regulations particularly at a time when, as a national industry, the pulp and paper industry is expanding its papermaking capacity at a faster rate than it is expanding its ability to produce pulp. It simply must be a national priority to encourage recycling.

The Wisconsin Paper Council recognizes that there is a potential problem with the introduction of PCB's into the environment. We strongly support strict controls on the manufacture, distribution, and use of PCB's and we favor a national ban on the importation of PCB's. But the paper industry has not created this problem, it does not use PCB's in its processes, and it finds itself caught in the middle of this controversy merely because of its recycling efforts, which would be halted by the adoption of overly restrictive regulations.

For example, proposed regulations in Wisconsin place an incredible burden on our industry, requiring removal of PCB's to levels 1,000 times smaller than the Food & Drug Administration's temporary tolerances as applied to food.

Our industry has a history of cooperation with government agencies to reduce PCB levels in our finished products. We will continue to cooperate with the Food & Drug Administration, with the EPA, and with our own DNR to find solutions to the PCB problem in effluent. We concurred with Mr. Schweitzer when he testified in Wisconsin that "it is important to clarify the portion of the PCB contamination problem which can be attributed to specific discharges and the *practical feasibility of reducing the discharges*." We hope that his Department recognizes that there is a difference in terms of environmental acceptance of various forms of PCB's and that this should be taken into account when dealing with any proposed regulations.

But we continue to appeal to you for reason. Let us admit together that we do not know enough, at least at this point, to promulgate reasonable regulations and that ill-conceived rules could mean economic catastrophe for a significant portion of Wisconsin's paper industry. The commitment of this industry is to Wisconsin and to its environmentally pure future. It has invested and will invest over $300 million prior to 1980 to accomplish that goal. We only hope that realistic, reasonable, feasible, and responsible regulations will be proposed by the EPA which will bolster its reputation as a defender of the environment while at the same time not place it in a position of ridicule because it has suggested regulations so badly researched that they are either unenforceable or that they destroy recycling—one of the this country's most environmentally considerate industries.

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CHAIRMAN TIMM. Now for another change of pace, David Kotelchuck, followed by Lee Botts.

DR. DAVID KOTELCHUCK (United Electrical Workers Union, New York, New York): At present, several States are proposing new PCB standards while EPA is actively considering such a standard. New standards for PCB's are clearly in process. I would like to discuss a matter that is of great importance to electrical workers, the people first affected by PCB's in the environment and the people whose lives are most immediately affected by the changes in the standards, both in terms of the health and jobs.

We want to point out that attempts to regulate PCB's use State by State leaves workers in existing plants subject to job loss through plants moving. United Electrical Workers represents over 1,000 workers at GE Capacitor plants at Fort Edwards and Hudson Falls, New York. Presently a New York State law is being considered phasing out PCB by next year. If the New York State laws pass in the absence of a Federal standard, then workers in New York and other States with stringent standards face possible plant moves and subsequent job loss.

Local GE plant officials have already publicly threatened to move if proposed New York State standards pass. We recognize the need for change in PCB standards to protect the health of workers and the general public. But we insist that this must be done by promulgating a Federal standard so as not to subject workers to this threat and not prejudice their job security relative to workers in other States.

It is incumbent upon EPA to set a new Federal standard. We want to point out that this is also the most effective way to regulate PCB in the environment.

One further point. We commend scientists who have examined health and environmental hazards of PCB. But a report of the results in medical and scientific literature does not end the scientist's responsiblility in our opinion. We believe that scientists also have a responsibility to inform electrical workers, those most directly affected by any PCB health hazard, of their results. In the past several years neither workers at the Fort Edward and Hudson Falls plants nor their national Union were informed by scientists or government officials of their findings or proposals. One cannot claim to be helping mankind by researching PCB hazards and regulating its use while ignoring precisely those individuals most intimately affected by PCB.

We request that as new scientific studies are conducted the authors please inform us of their results. Our address: Research Department, United Electrical Workers, 11 East 51st Street, New York, New York 10022. Thank you.

BETTER LATE THAN NEVER: THE CASE FOR TREATING PCB'S AS TOXIC SUBSTANCES NOW

Lee Botts*

My name is Lee Botts, and I represent the Lake Michigan Federation, a coalition of citizen groups and individuals with members in Wisconsin, Illinois, Michigan, and Indiana. Our affilitates include organizations of conservationists, of sports fishermen, of organized labor, of school children, and of people who could be classified under many other labels but share a common concern for protection of Lake Michigan from manmade pollution. Today I am here to express the dismay of our organization over the failure of the Government of the United States to protect the lake and those of us who depend on it for drinking water, for food, and for the quality of our lives in the future against contamination by polychlorinated biphenyls (PCB's).

The fact that such contimaination exists is not news. Attention was called to PCB's as a potential danger in a Lake Michigan Enforcement Conference in 1971. We worked for and celebrated the passage of the Water Pollution Control Act amendments in 1972. We have asked for and waited for regulatory action on toxic substances that would include PCB's as provided for under Section 307. In the past year we have become increasingly alarmed over the failure of EPA to make use of its powers under Section 504.

Under this section, the administrator may bring suit on behalf of the United States to stop pollution that threatens either the public health or the means of livelihood of people. PCB's do both, and yet what has EPA done?

EPA ACTIONS TO DATE

Since 1972, EPA officials have been assuring themselves, if not the rest of us, that, in the words of John Buckley, chairman of this conference, "The PCB problem is fairly well in hand." This statement was attributed to him in a *Science* magazine discussion of PCB's in October, 1972 (page 388, Vol. 178). Just how EPA reached this conclusion is a mystery, since it was not confirmed by the monitoring efforts being made by agencies here in Region V, including the National Water Quality Laboratory in Duluth and the Great Lakes Fishery Laboratory in Ann Arbor.

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*Executive Director, Lake Michigan Federation, Chicago, Illinois.

When confronted with the rising levels of PCB's found by monitoring in the Great Lakes, Washington EPA officials dismissed the problem as a regional one. Now this is a fairly large region, and it might have seemed reasonable to investigate whether PCB's were present in other waterways when their existence in the largest fresh water system in the world was so widespread. But evidently the thought was that if you do not look for a problem, you will not have to deal with it. Unfortunately, the problem was not only present elsewhere; research confirmed that it was growing worse.

NATURE OF THE PCB PROBLEMS

In various research efforts, the nature of the problem was becoming clear. First, there is the fact that PCB's have high concentration factors, considerably higher than those of the chlorinated hydrocarbon pesticides. Evidence suggests that PCB's may not be as soluble in water, but there is no doubt now that biomagnification of PCB's in the food chain is very great.

Second, a number of the 190 different PCB compounds degrade very slowly. The U.S. Department of Agriculture found in experiments reported in a 1972 conference that the isomers in the intermediate range of chlorine content were more toxic to chickens than those with either very small or very high chlorine content.

Third, it was learned that, like DDT and other pesticides, PCB's are stored in fatty tissues of animals and man, ready to be released with metabolism of the fat. One 1974 report on this phenomenon in the Bulletin of Environmental Control and Technology found that normal persons seem to have small blood level concentrations of both DDT and PCB's even if they have higher levels in stored fat. But nine persons with cancer were found to have extremely high levels of both the degraded form of DDT and PCB's in their blood, suggesting that the toxic chemicals had been released into the bloodstream during an illness that causes weight loss through fat metabolism. The authors of this report speculate that this mechanism may offer a much more serious long-term threat to health than was previously realized, since it would affect people already sick, or growing old, or even those trying to improve their health by dieting to lose weight.

The point is that for several years many investigators have been trying to find out not only whether PCB's are toxic but how they cause trouble. That is, many investigators outside EPA were studying the problem.

WHAT EPA HAS NOT DONE

In this connection, let us consider what EPA has not done that might have reasonably been expected of the agency that has the charge of protecting the environment and public health in general but has the power to regulate specific toxic substances.

The major action to determine sources of PCB's was to request information from industries that might have occasion to discharge wastes containing PCB's into waterways, that is, to be point sources of PCB's. Since Section 308 applies only to owners and operators of such sources, this inquiry was not adequate to disclose all the industrial users of PCB's nor means of distribution from manufacturers through middlemen to actual users. Strictly speaking, an industry that did not discharge PCB's directly would not have to provide information under Section 308 letters about whether it made and sold products containing the chemicals that might ultimately reach the environment by other means.

Conceivably, EPA might have been trying to find out for itself just how the PCB's were being scattered so widely. But EPA did not, for example, investigate whether PCB's are finding their way into rivers and lakes from sanitary landfills. Nor did it obtain information about possible atmospheric transfer, even though the fact that other substances are carried into bodies of water like Lake Michigan by rainfall is stimulating attention to this mechanism.

Having failed to take advantage of PL 92-500, it might have been supposed that EPA would turn to other means of regulation. Again, not so. PCB's are not regulated under the Safe Drinking Water Act, either.

CONCLUSION

I spoke in the beginning of the failure of the Federal Government to deal adequately with the PCB problem, but in truth some agencies have already taken some initiative. The Food and Drug Administration is amending its food packaging regulations to prevent contamination by plastic wrappings. The Department of Defense and the General Service Administration have indicated willingness to restrict purchase and use of items containing or using PCB's.

Some States have acted, at least to protect the public even though such actions can do little to prevent continuing environmental contamination. Wisconsin and Michigan have issued warnings to the public about eating fish caught in Lake Michigan more than once a week. This warning, of course, is cold comfort indeed to the fishermen, whether for sport or for a living. Present at this meeting are representatives of both groups who will speak to their own interests in this matter.

In calling for decisive regulatory action by the Environmental Protection Agency now, I would like to submit to you several petitions signed by individual members of one of the member organizations of the Lake Michigan Federation, the National Council of Jewish Women. This is tangible evidence of their concern, but I can assert with confidence that it is shared not only by other members of the Lake Michigan Federation but almost everyone who understands the nature of this problem. EPA must understand that its leadership is needed to deal with this national problem now.

A FAILURE OF GOVERNMENT

Richard R. Knabel*

Representing:
The Hudson River Fisherman's Association
The Hudson River Sloop Restoration
The Federated Conservationists of Westchester County

The Hudson River Fisherman's Association, the Sloop Restoration, and the Federated Conservationists of Westchester County together represent the major elements of the environmental movement in the Hudson Valley. They have collectively helped to define many of the environmental safeguards, such as they are, that have come into being since public awareness of ecological problems came into its own during the past 10 or 12 years.

My comments today, on behalf of these groups, are intended to raise the "decibel level," as Administrator Train commented yesterday, but not intended to criticize the efforts of this conference or its participants. What we in the Hudson Valley have experienced over the past 3 months, and all that I have heard since arriving in Chicago, indicates that the decibel level has not yet reached the pain threshold in government offices. We are astonished at the delays. We cannot understand why it has taken so long for the PCB menace to surface, and we are horrified that so little action seems to be contemplated. We wonder what would have happened if the exposé begun by Commissioner of Environmental Conservation, Ogden Reid, in New York State had not occurred.

As many of you may know, this conference owes its genesis, in part, to the almost unbelievable revelations in the Hudson Valley last August. On August 8, 1975, Commissioner Reid dropped a bombshell on the public by warning them not to eat striped bass from both the Hudson River and Lake Ontario because data given him by the EPA indicated levels of PCB's exceeding 5 ppm were found in the fish taken from both waterbodies. He identified the culprit in the case of the Hudson River as primarily the two General Electric facilities at Fort Edward and Hudson Falls, New York, whose massive capacitor plants there were dumping an average of 30 lb. per day of PCB. The river is apparently contaminated for a distance exceeding 100 miles, and the entire fishery is in question.

As one might imagine, the reaction to this stunning announcement was one of shock, frustration, disgust, dismay and confusion. The reasonably successful cleanup of sewage and industrial waste in the Hudson Valley, while far from complete, was making visible progress. The water was not only cleaner to the eye, but the increased variety of fish returning to the river in numbers not seen by commercial fishermen for over 20 years inspired confidence and pride. Since 1972, the Sloop Restoration had been sponsoring an annual shad sail in which thousands of people had participated by eating freshly caught shad. Eating fish from a cleaner Hudson was a natural outgrowth of the cleaner water we all thought existed. Imagine the concern, and fright of all these people on the morning of August 8th.

When the shock wore off somewhat, many of us asked; was this a new discovery? Was this just another layer of contamination that no one had looked for before? Did new analytical techniques, or a fortuitous investigation produce this damaging information? To our horror, consternation, and anger, what we found, in New York State anyway, was an Environmental Watergate that knows no parallel. The confusion rapidly changed to an angry feeling of betrayal.

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*Director, Hudson River Fishermen's Association, Yorktown Heights, New York.

As early as February 17, 1971, we discovered, efforts were made by Mr. Robert Boyle of *Sports Illustrated*, and perhaps others, to alert New York State environmental officials, notably Mr. Carl Parker, chief of the Bureau of Fisheries, to the presence of dangerously high PCB levels in striped bass eggs, and levels approaching the cutoff of 5.0 ppm in fish tissue. Mr. Boyle describes the response he got as "derisive." Not only was no action taken by State officials, but considerable evidence exists that active efforts to suppress any information about PCB contamination persisted within the department for several years. All of this despite reports of the problem written by Boyle in both *Sports Illustrated* and *Audubon Magazine* in 1970.

In June, 1971, the New York State Department of Health certified that the water quality of the discharge from the two enormous General Electric plants met its criteria. In spite of the fact that 5.7 million lb of PCB's are used at these plants each year, no mention of PCB's was made by the health department.

In July and through September 1972 (1½ years after Boyle first signaled trouble), New York State undertook PCB analyses in the mid-Hudson area and discovered concentrations exceeding FDA limits of 5.0 ppm in fish. This information never saw the light of day. The then Commissioner, Henry Diamond, recently disclaimed any knowledge of the study or its damaging results. His successor, James Biggane, also denies knowledge, and the new Commissioner claims he found out from Federal sources, not from within his own department. Reid said in a *New York Times* story, by Richard Savero, "There has been a failure of government here...," referring to the suppression of information within his newly aquired department. That was a masterful example of understatement.

Subsequent further investigation revealed that between 1970 and 1975, 99 analyses of Hudson River fish were conducted by State officials, and in 52 cases the PCB levels were greater than the maximum allowable level of 5.0 ppm. A largemouth bass caught at the mouth of Esopus Creek, about 80 miles north of New York City, in 1970, but not analyzed until 1972, contained 53.8 ppm, or more than 10 times the maximum allowable limit. Subsequently, levels were detected in several species that ranged to over 100 ppm, with levels generally decreasing as distance from the GE discharges increased.

On September 8, 1975, GE was ordered by the State to stop dumping its average of 30 lb per day by September 30, 1976, and to immediately curtail its discharges to 2 lb per day as of September 30, 1975. Reid required GE to post a $2 million performance bond to back up his order. GE's response was to contest the order, as it did the provisions of its NPDES permit, which also required a major curtailment of PCB discharges, and to claim that it had already voluntarily reduced the discharge levels to under 2 lb per day. However, in the course of hearings, going on at this moment in Albany, it has been revealed that in the 24-hour period between October 17 and 18, 1975, 75 lb of PCB's were dumped at the Fort Edward Plant, by virtue of "heavy rain during sewer line repair." On September 13-14, 1975, a horrendous 116 lb of PCB's were dumped by the Hudson Falls plant with no reason stated. Both pieces of data came from GE's own written responses to questions raised by State environmental lawyers. Based upon data the EPA has presented at this meeting, both of these occurances represent major spills. I wonder if the EPA is aware of them, and what they will do about them.

In any case, it is clear that GE has not curtailed its discharges, and it seems probable that this random and massive dosing of the Hudson will continue to poison and jeopardize the health, safety, and welfare of residents not only in the valley, but all along the northeastern seaboard, where commercial fishermen catch fish born and raised in the Hudson Estuary.

While the record of State officials is both shameful and scandalous, the EPA has not acted with openness, or alacrity either. I should say that Region II's behavior has left much to be desired in this matter. Without providing the full details, which I will gladly do for anyone desiring them, our investigations of the Region II office's actions indicate that it too had knowledge of the severity and extent of the PCB problem in the Hudson long before it forwarded the data to Albany or made it public themselves.

An internal EPA memo dated May 31, 1974, reveals a request from Sandra Kunsberg, attorney in the Water Enforcement Branch, to Dr. Richard Spears, Chief of Surveillance and Analysis Division, to conduct sampling at the GE plants and in the Hudson River, to "Determine whether or not there exists a health hazard within the meaning of Section 504 of the Water Pollution Control Act." This request was occasioned by testimony at the May 1974 EPA hearings on toxic substances standards where GE revealed the extent of its PCB discharges into the Hudson River.

The EPA lab report on this investigation, dated October 1974, concluded "that the biotic component of the river ecology are heavily contaminated with PCB's, grazing populations as well as carnivorous piscine populations," and notes that certain areas around Fort Edward and Hudson Falls "are fished primarily by the youngsters of Fort Edward. Ingestion of these fish by the populus would certainly lead to contamination of specific tissues in their bodies...." There was no health alert. No news release. There was nothing.

Almost 1 year went by before this information finally reached the new environmental commissioner of New York. The question again is why? We must ask, how can so-called public servants walk around with the knowledge that commercial and sports fishermen up and down the North Atlantic as well as on the Hudson were catching Hudson River striped bass, shad, small mouth bass, and eels for human consumption, knowing full well that the likelihood of their contamination was great, that the fish posed a health hazard, and that their oaths of office required action. That this situation could persist for literally years is unforgiveable. A failure of government? No. Indictable criminal offenses more suited to investigation by a grand jury than discussion at a conference such as this one.

As far as action to end the problem nationwide is concerned, we decry the procrastination, fear, or whatever, that seems to underlie the apparent lack of direction and assertiveness thus far displayed. There still appears to be a question about what comes first, the national health or the business interests of the electrical and other industries that rely upon PCB's for their profits.

We fail to see how a proposed standard of 1 part per trillion ambient in the Nation's waterways will solve the problem. Is it low enough? Can it be enforced? How was it arrived at? Can we live with PCB's at all? What will happen, or rather what should happen to the more than 100 million lb of PCB still available to be degraded or released into the environment? How will we deal with this problem?

It is equally alarming to note that the FDA has not stated here one syllable about any proposed change in the permissible PCB levels in food or fish. We wonder why fish have highest permissible PCB level in the first place. The suggestion that PCB levels are decreasing in all foods except fish seems to contradict the ubiquitous nature of PCB's, and creates a false sense of security. Our Canadian neighbors have announced a new lower standard of 2.0 ppm in food, and Commissioner Ogden Reid has taken the position, to his credit again, that a standard of 1.0 ppm seems to be desirable. When will the FDA take some action?

Getting back to the Hudson Valley, which we hope is not the average situation around the country, the fledgling Hudson River fishery, thanks to GE, is dead for all intents and purposes. Commercial fishermen who had hoped to go back to fishing full time have scrapped their plans. Even if their catches were not contaminated, fish markets won't buy them or pay such low prices that no profit is possible. The public is now convinced that Hudson fish are tainted, and will continue to think so for many years regardless of cleanup requirements or measurable improvements that may or may not take place. The implications for the North Atlantic fishery have yet to be explored or defined, but the potential economic impact of the August 8th revelation, an environmental Pearl Harbour, for fishermen everywhere is devastating. Confidence in government and in the Hudson's recovery is at a new low.

In conclusion, the time to act is now. Administrator Train said yesterday that he hopes this will be his last PCB conference. Well, we also hope that no more conferences on PCB's will be necessary. How much data does it take before action can be justified? Must we suffer an outbreak of Yusho disease before the problem assumes real proportions? We must have more than conferences. And we expect more than talk.

Thank you for your attention, and for this opportunity to speak.

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MS. EILEEN JOHNSTON (Concerned citizen): Thank you, Chris. Well, I would like to thank the U.S. Environmental Protection Agency for putting on this stimulating meeting. I felt privileged to be in the room with so many fantastic research members and I was a little encouraged and discouraged. I happen to live in the Lake Michigan basin. I was wondering, too, where are all the concerned citizens. I really saw very few people from Illinois that I felt should have been here if they were concerned about PCB's.

I also want to congratulate the State of Wisconsin for the hearings that they have held, and I'll be very anxious to see what comes from those, and also to congratulate the State of Michigan for the fine presentation that John Hesse made. I will be interested to follow House Bill 5619 in Michigan.

My own great concern for PCB's in the environment is due to my own Illinois Environmental Protection Agency. Last February the agency was asked to host the February meeting for the Governor's five-State Interdisciplinary Council on Pesticides. Jim Frank, a very capable young man from EPA, put on an all-day meeting on PCB's. I was the only citizen at this meeting and it was so stimulating that I started out to do what an individual citizen can do. I talked to a few citizen groups that I am associated with and we decided to put on a meeting to help warn the Lake Michigan fishermen about this and we held the meeting in March or April.

I urged more citizen groups to do this sort of thing—to take positive action. Citizens need more education. I hope that USEPA and everybody else concerned with PCB's will really listen to Carlos Fetterholf, who really socked it to people tonight.

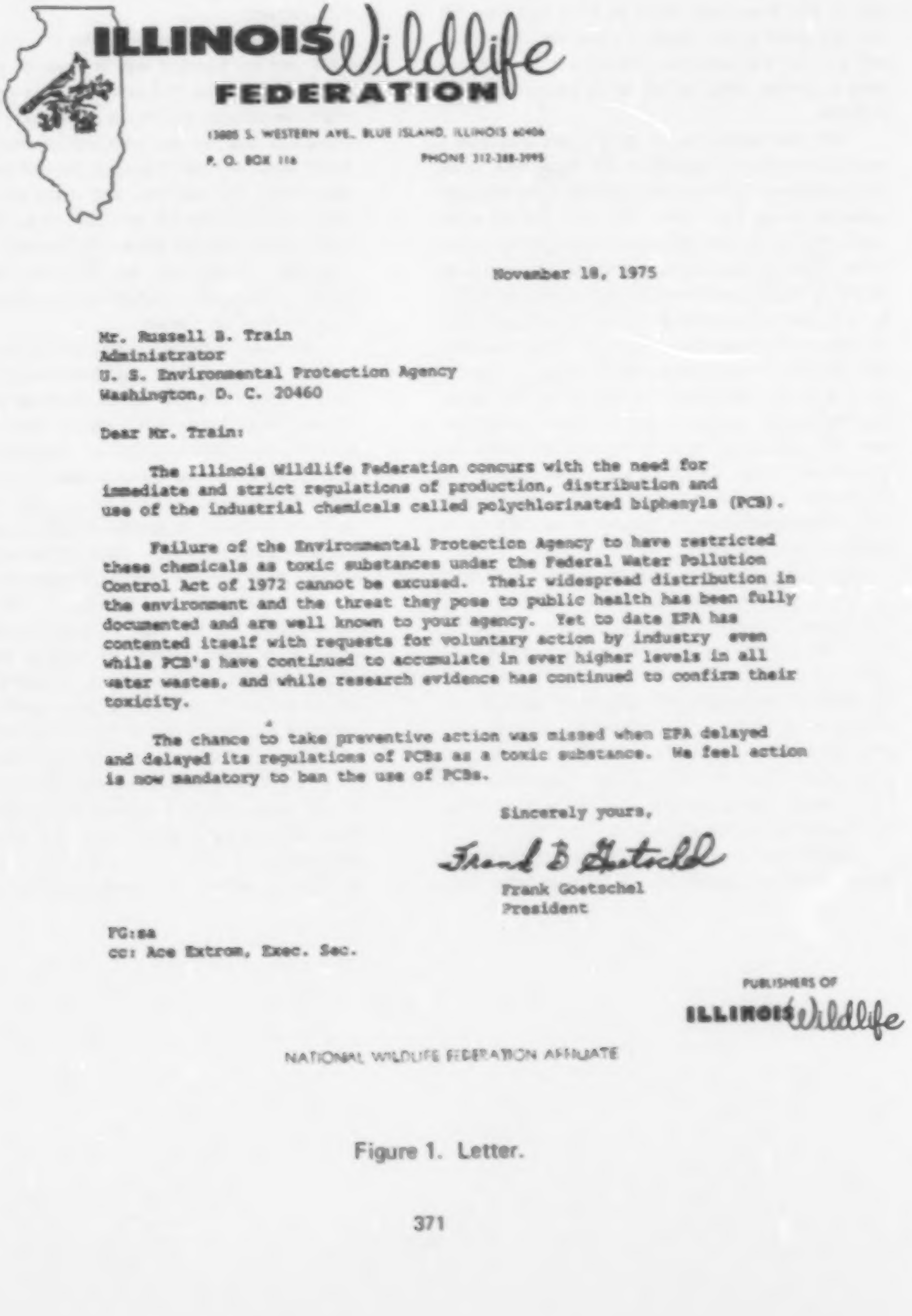
Getting back to Illinois, I've talked to 225 citizens quite recently and explained to them the perils of PCB's and they signed petitions for me to take to the Illinois Pollution Control Board to ask it to hold informational hearings on PCB's, to get them informed, and to get our own Illinois EPA and interested citizens to be better informed. They decided to wait until after these meetings to decide whether to hold hearings.

Another thing I heard today that the researchers mentioned is that we ought to get together and get some standardized analytical methods. Remember when we were concerned about DDT? I attended a 2-day seminar out on Pershing Road at EPA headquarters. Everybody was analyzing whole fish, half fish, and it seems to me from what I heard that they really should get together and develop uniform procedures.

I think it was a great conference but I really want to know what EPA is going to do. What are you guys going to do? I have great faith in the U.S. Environmental Protection Agency. I know so many people in Region V—but what are you going to do? Are you going to Congress? Are you going to take action? Are you going to answer the questions tonight, Chris? Thank you.

Session continued on next page.

MR. RAY OLTMANNS (Illinois Wildlife Federation, Blue Island, Illinois): I am a delegate from the Illinois Wildlife Federation. I am going to read a letter (figure 1) that is going to go to Mr. Train; it is from Frank Goetschel, our president. Thank you.



ILLINOIS Wildlife
FEDERATION
13800 S. WESTERN AVE., BLUE ISLAND, ILLINOIS 60406
P. O. BOX 116
PHONE 312-388-3995

November 18, 1975

Mr. Russell B. Train
Administrator
U. S. Environmental Protection Agency
Washington, D. C. 20460

Dear Mr. Train:

The Illinois Wildlife Federation concurs with the need for immediate and strict regulations of production, distribution and use of the industrial chemicals called polychlorinated biphenyls (PCB).

Failure of the Environmental Protection Agency to have restricted these chemicals as toxic substances under the Federal Water Pollution Control Act of 1972 cannot be excused. Their widespread distribution in the environment and the threat they pose to public health has been fully documented and are well known to your agency. Yet to date EPA has contented itself with requests for voluntary action by industry even while PCB's have continued to accumulate in ever higher levels in all water wastes, and while research evidence has continued to confirm their toxicity.

The chance to take preventive action was missed when EPA delayed and delayed its regulations of PCBs as a toxic substance. We feel action is now mandatory to ban the use of PCBs.

Sincerely yours,

Frank B Goetschel
Frank Goetschel
President

FG:sa
cc: Ace Extrom, Exec. Sec.

PUBLISHERS OF
ILLINOIS Wildlife

NATIONAL WILDLIFE FEDERATION AFFILIATE

Figure 1. Letter.

MR. BURNETT BAUER (State Senator from South Bend, Indiana): First of all, I want to congratulate the EPA for holding a meeting at which it can't really win. I've heard in this meeting that they've done too much and then I've heard them damned for doing too little. And they don't have to do that; we in politics every election have to stand up and take it, but they really don't have to. But they did call this meeting and I think it's been very beneficial and the fact that we've had discussions is a real step toward solving what all of us recognize is a real problem.

Now the reason I want to talk here is because I want to make one suggestion for those who want the government to take some action. That was suggested a number of times. It'll also maybe warn those who don't want the government to do a darn thing. What I'm suggesting is this, that you not wait for the Federal Government to start action on PCB's but you start it in the States. I know that's contrary to most of the procedure and most of the thoughts that people have, particularly those in State Government. But the historical fact is most of the social changes in our country started in States first. The women's sufferage movement was not suddenly brought on by the Federal Government; the States first started allowing women to vote. We know that most of the environmental actions were started in a locality and in the State.

It's just a fact that once the Federal Government sees States acting that this indicates to them seriousness, and then they go in and invest the time and money that they have. Now there are several reasons why we want the States to do this. First of all, most of the legislatures are citizen legislatures. In one sense, we're not as vulnerable to lobbyists. Most of us are serving there because we want to better our State. We want to give our children a better place in which to live, and we are doing what we think primarily would be best in the long run.

Second, we are closer to specific problems and when we put a solution in there, it is probably a lot closer to a genuine solution. It's much harder for people in Washington to try to legislate something that applies over the 50 States. If you start with your actions at home—and God bless these women out here who have these groups, they'll bring you specific instances you'll start legislating laws that will work. I'll give you just ,one instance and then I'll sit down.

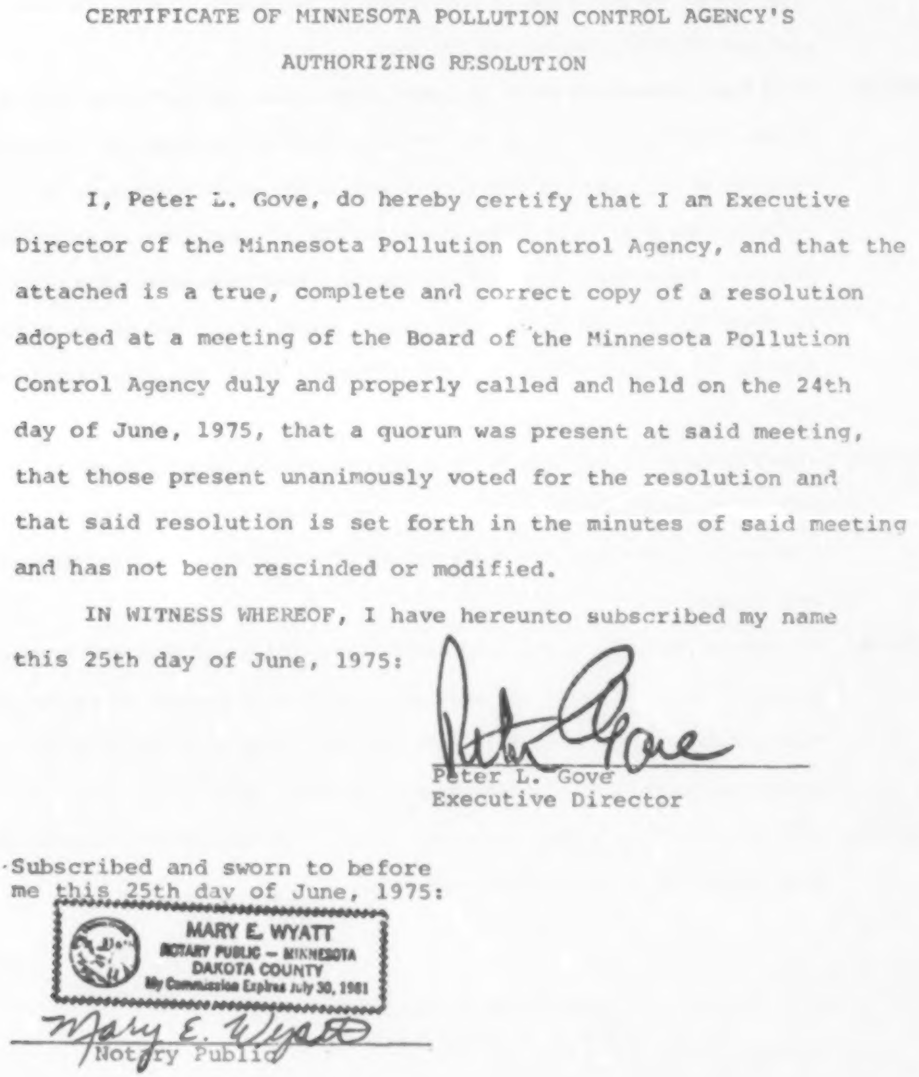
In Indiana we have pollution of a lot of our fine lakes and we heard it was because of phosphates. Indiana passed the first statewide ban against phosphates in detergents. I'm the grandfather of that bill because it was my son, who just happened to be in the House, who got it passed. He had more energy than I did. The real fact was that I was out for 2 years and the lobbyists went out of the State. They didn't think the bill would be brought up. By the time they found out, the Governor had already signed it. So there's something in passing the work onto the next generation.

We have found as a result of that ban that our State's lakes are clearing up fantastically. That law works. New York now has it. We hope other States of the Great Lakes will adopt it. One reason I'm here is I have met with groups that are around the Great Lakes and the Great Lakes are in danger of many things. And I just want to urge you people who are in favor of having something done to go home and start working on your State legislatures. I know I'll get some letters from some of those guys saying, "why don't you shut up, Bauer, we've got enough work to do." But do that if you really are genuine in your interest of getting some action toward cleaning up our environment, and in passing on to the next generation an atmosphere and environment that can make us happy, and can make us really enjoy the greatest country in the world.

I want to congratulate again EPA and every one of you who came here from industry. I've learned a heck of a lot here, and I think that all those who participated in this meeting are really leaders and genuine pioneers in this country. I salute you.

MR. BARRY SCHADE (Minnesota Pollution Control Agency, Roseville, Minnesota): The Minnesota Pollution Control Agency is a member of a task force made up of 11 agencies which represent the various interests of two States and the Federal Government.

This task force was formed to investigate the PCB pollution problem in the upper Mississippi River and its tributaries. In response to preliminary information provided by this task force and by various other studies, the Minnesota Pollution Control Agency board passed a resolution related to PCB's on June 24th, 1975. This resolution, which supports a Federal ban on the sale and use of PCB's, is submitted as figure 1.



CERTIFICATE OF MINNESOTA POLLUTION CONTROL AGENCY'S
AUTHORIZING RESOLUTION

I, Peter L. Gove, do hereby certify that I am Executive Director of the Minnesota Pollution Control Agency, and that the attached is a true, complete and correct copy of a resolution adopted at a meeting of the Board of the Minnesota Pollution Control Agency duly and properly called and held on the 24th day of June, 1975, that a quorum was present at said meeting, that those present unanimously voted for the resolution and that said resolution is set forth in the minutes of said meeting and has not been rescinded or modified.

IN WITNESS WHEREOF, I have hereunto subscribed my name this 25th day of June, 1975:

Peter Gove

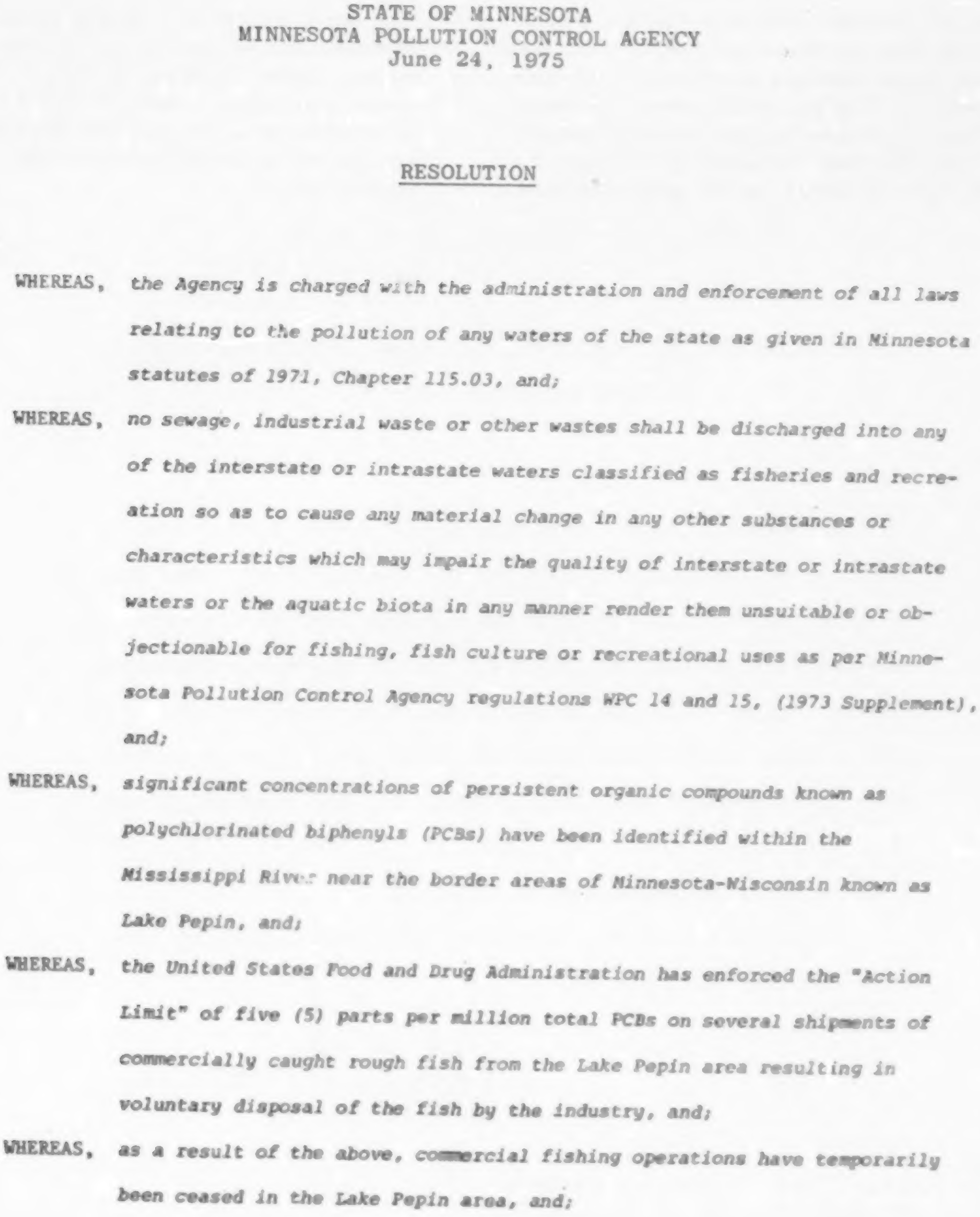
Peter L. Gove
Executive Director

Subscribed and sworn to before
me this 25th day of June, 1975:

MARY E. WYATT
NOTARY PUBLIC — MINNESOTA
DAKOTA COUNTY
My Commission Expires July 30, 1981

Mary E. Wyatt
Notary Public

Figure 1. Resolution of Minnesota Pollution Control Board.



STATE OF MINNESOTA
MINNESOTA POLLUTION CONTROL AGENCY
June 24, 1975

RESOLUTION

WHEREAS, *the Agency is charged with the administration and enforcement of all laws relating to the pollution of any waters of the state as given in Minnesota statutes of 1971, Chapter 115.03, and;*

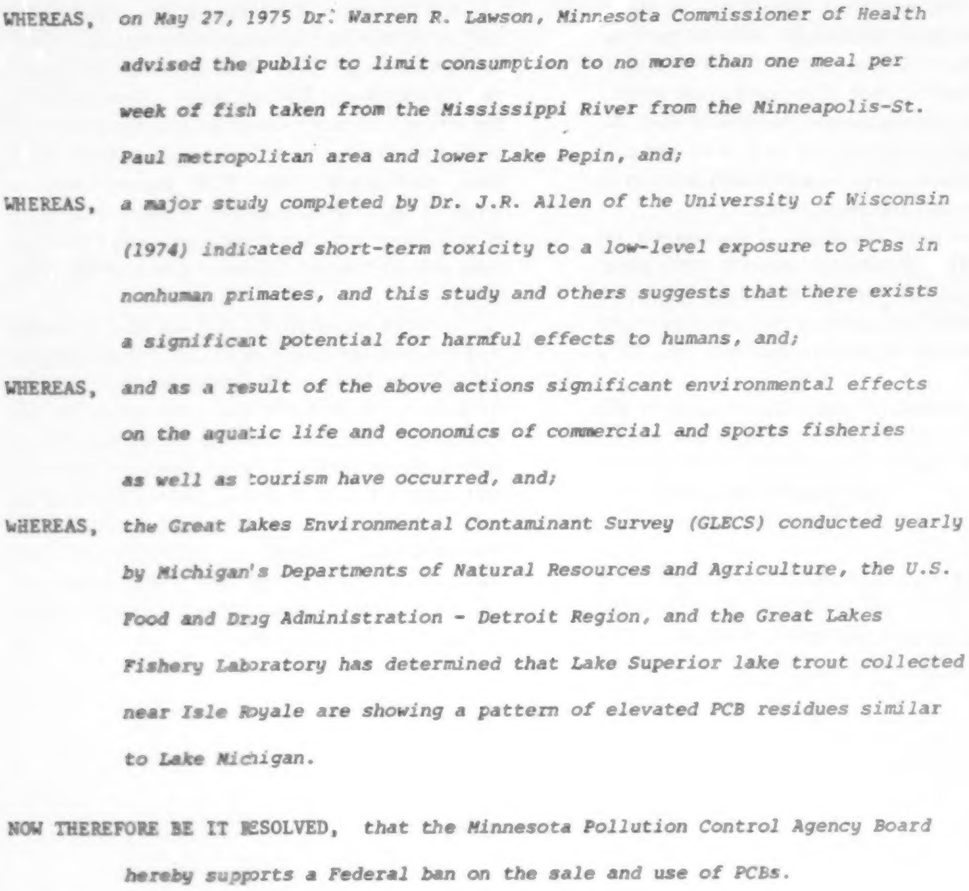
WHEREAS, *no sewage, industrial waste or other wastes shall be discharged into any of the interstate or intrastate waters classified as fisheries and recreation so as to cause any material change in any other substances or characteristics which may impair the quality of interstate or intrastate waters or the aquatic biota in any manner render them unsuitable or objectionable for fishing, fish culture or recreational uses as per Minnesota Pollution Control Agency regulations WPC 14 and 15, (1973 Supplement), and;*

WHEREAS, *significant concentrations of persistent organic compounds known as polychlorinated biphenyls (PCBs) have been identified within the Mississippi River near the border areas of Minnesota-Wisconsin known as Lake Pepin, and;*

WHEREAS, *the United States Food and Drug Administration has enforced the "Action Limit" of five (5) parts per million total PCBs on several shipments of commercially caught rough fish from the Lake Pepin area resulting in voluntary disposal of the fish by the industry, and;*

WHEREAS, *as a result of the above, commercial fishing operations have temporarily been ceased in the Lake Pepin area, and;*

Figure 1. Resolution of Minnesota Pollution Control Board (con.).



WHEREAS, *on May 27, 1975 Dr. Warren R. Lawson, Minnesota Commissioner of Health advised the public to limit consumption to no more than one meal per week of fish taken from the Mississippi River from the Minneapolis-St. Paul metropolitan area and lower Lake Pepin, and;*

WHEREAS, *a major study completed by Dr. J.R. Allen of the University of Wisconsin (1974) indicated short-term toxicity to a low-level exposure to PCBs in nonhuman primates, and this study and others suggests that there exists a significant potential for harmful effects to humans, and;*

WHEREAS, *and as a result of the above actions significant environmental effects on the aquatic life and economics of commercial and sports fisheries as well as tourism have occurred, and;*

WHEREAS, *the Great Lakes Environmental Contaminant Survey (GLECS) conducted yearly by Michigan's Departments of Natural Resources and Agriculture, the U.S. Food and Drug Administration - Detroit Region, and the Great Lakes Fishery Laboratory has determined that Lake Superior lake trout collected near Isle Royale are showing a pattern of elevated PCB residues similar to Lake Michigan.*

NOW THEREFORE BE IT RESOLVED, *that the Minnesota Pollution Control Agency Board hereby supports a Federal ban on the sale and use of PCBs.*

Figure 1. Resolution of Minnesota Pollution Control Board (con.).

MR. RICHARD T. FERRY (Bio-International, Inc., Woods Hole, Massachusetts): Bio-International, Inc., is principally located in Ft. Lauderdale, Florida, and holds four U.S. patents covering its processes for the microbial degradation of petroleum and petroleum byproducts. Bio-International holds one United States patent for a microbial degradation facility; that is, a mechanical facility comprising an interconnected initial and final degradation system, each containing an inlet and outlet and a conveying means together with aeration or stirring devices and a drainage means.

This patent was issued only 3 months ago, on August 11, 1975. Of particular interest to the group here today is a patent held by the company relating to the microbial degradation of polychlorinated biphenyls that was issued on December 18, 1973 (see figure 1).

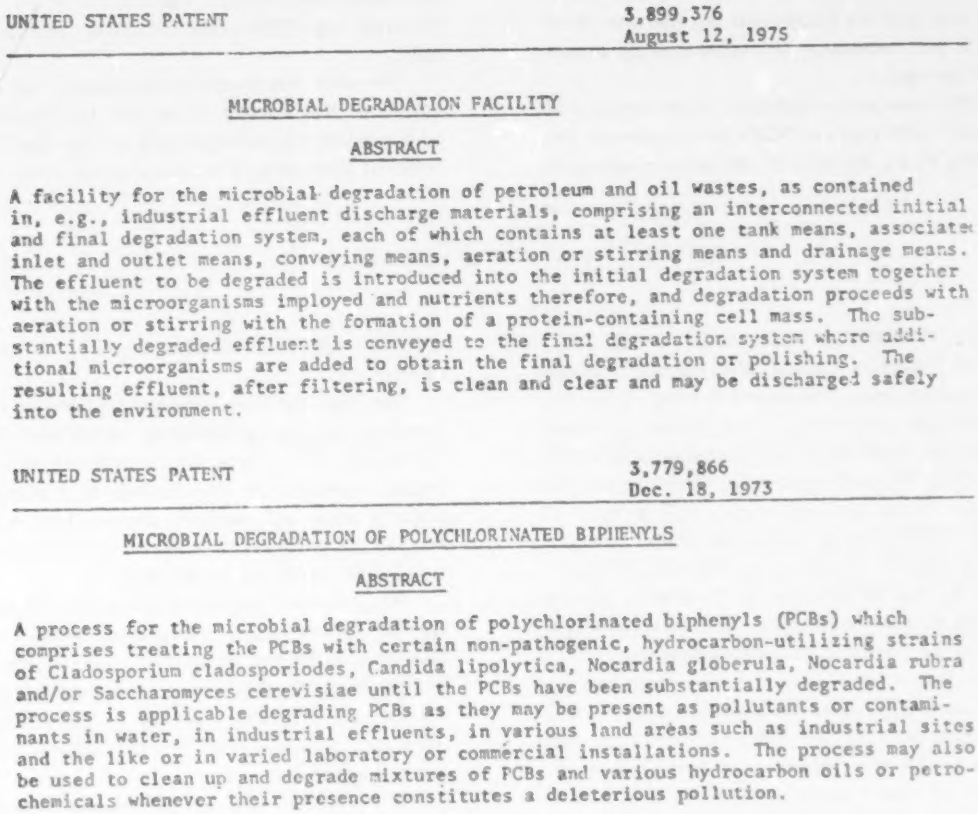
Bio-International is currently engaged in the optimization of its processes at three leading universities in the Southeast. The purpose of this ongoing work is to further increase the efficiency of the integrated system and to broaden the scope of field applications.

In the process, a significant amount of scientific and technical data is being electronically stored for quick retrieval when circumstances demand. This information is also being synthesized in order to prepare an affirmative response to the revised Annex Ten of the National Contingency Plan relative to the introduction of microbial additives to the environment.

Additionally, Bio-International maintains a staff of internationally recognized scientific authorities whose main thrust is in the area of environmental consulting. The company is now analyzing the effluent of major industries who recognize their own obligation to solve their own effluent problems, particularly where PCB contamination is involved. Upon the completion of these analyses, it is envisioned that the company's integrated processes will be brought to bear on the problem effluents.

I would be remiss if I did not note in closing that our ongoing studies relative to the problems of PCB's in the environment would be much more difficult were it not for the cooperation of Mr. Papageorge and his colleagues at Monsanto as well as other representatives of government and industry, particularly the fishing and marine-related industries. For those of you who may be interested in the biodegradation approach to environmental problems, you're invited to contact me here or by letter and we'll be happy to provide you with more specific data. Thank you very much.

Figure 1 on following page.



UNITED STATES PATENT

3,899,376
August 12, 1975

MICROBIAL DEGRADATION FACILITY

ABSTRACT

A facility for the microbial degradation of petroleum and oil wastes, as contained in, e.g., industrial effluent discharge materials, comprising an interconnected initial and final degradation system, each of which contains at least one tank means, associated inlet and outlet means, conveying means, aeration or stirring means and drainage means. The effluent to be degraded is introduced into the initial degradation system together with the microorganisms imployed and nutrients therefore, and degradation proceeds with aeration or stirring with the formation of a protein-containing cell mass. The substantially degraded effluent is conveyed to the final degradation system where additional microorganisms are added to obtain the final degradation or polishing. The resulting effluent, after filtering, is clean and clear and may be discharged safely into the environment.

UNITED STATES PATENT

3,779,866
Dec. 18, 1973

MICROBIAL DEGRADATION OF POLYCHLORINATED BIPHENYLS

ABSTRACT

A process for the microbial degradation of polychlorinated biphenyls (PCBs) which comprises treating the PCBs with certain non-pathogenic, hydrocarbon-utilizing strains of Cladosporium cladosporiodes, Candida lipolytica, Nocardia globerula, Nocardia rubra and/or Saccharomyces cerevisiae until the PCBs have been substantially degraded. The process is applicable degrading PCBs as they may be present as pollutants or contaminants in water, in industrial effluents, in various land areas such as industrial sites and the like or in varied laboratory or commercial installations. The process may also be used to clean up and degrade mixtures of PCBs and various hydrocarbon oils or petrochemicals whenever their presence constitutes a deleterious pollution.

Figure 1. Abstract of patent relating to the microbial degradation of polychlorinated biphenyls.

* * * * * * * * * * * * * * * * *

MAJOR GORDON GOFF (U.S. Army Materiel Command, Alexandria, Virginia): The following description of the spill of 265 gallons of PCB's into the Duwamish Waterway in Seattle is presented here as an ongoing practical problem that we're facing in the recovery and disposal of contaminated bottom sediments from a busy waterway in this industrial area.

The spill itself occurred on September 13, 1974, when a large electrical transformer fully sheath-crated for open-deck shipment to Alaska was being sling-loaded aboard a barge. As the slung crate was about 2 feet off the dock the bottom members gave way, the transformer fell to the dock, and the outboard cooling fins struck the bull rail, rupturing them and leaking approximately 265 gallons of PCB material in the transformer onto the dock and into the waterway.

At that point in time, because of a lack of awareness of the people on the scene as to exactly what was in the transformer and the lack of labeling or warning on the crating itself, the normal oil spill procedures used at that dock were instituted. Within 2 or 3 days, however, the possibility that PCB's were implicated in the spill appeared. Subsequent investigations bore out the fact that the spill was indeed PCB's. EPA and the State of Washington Department of Ecology instituted an emergency cleanup procedure. Handheld dredges manned by divers recovered the visible pools of PCB's which were lying on the bottom of the waterway.

A filtration unit was then moved into the area and the recovered material was run through this filtration unit and the sludge that resulted was drummed and was subsequently disposed of by a commercial contractor.

In this emergency operation, approximately 80 gallons of visible pools of PCB's were recovered. The remaining PCB's are now in the bottom sediments and subsequent sampling has established an area of relatively high PCB contamination in the area of the spill.

To cover the proposed procedures for the recovery of this I'd like to ask Mr. Jack Thompson, who is a fisheries biologist with the Seattle District Corps of Engineers, to cover the details of it.

* * * * * * * * * * * * * * * *

MR. JOHN S. THOMPSON (Seattle District, U.S. Army Corps of Engineers, Seattle, Washington): The previous speaker described events leading up to the accidental loss of PCB's into the Duwamish Waterway, Seattle, Washington, and partial removal of the PCB's by EPA and Washington Department of Ecology. There remains about 185 gallons of Aroclor 1242 in the immediate area where the spill occurred. About 2 months ago the Corps was requested by the Department of Defense to remove the contaminated sediments. This decision is supported by EPA and the State of Washington.

The Duwamish Waterway is a dredged channel in the Duwamish River. It is dredged a distance of 5 miles from salt water with one-half of the channel dredged to 30 feet below mean lower low water (MLLW). The spill occurred at about river mile 2, where the dredged channel is 30 feet below mean lower low water.

Background levels of PCB's in bottom sediments run from 0.16 ppm to about 2.5 ppm. Levels of PCB's in the area of spill range from about 5.4 ppm up to 390 ppm in the sediments. We propose to remove the contaminated sediments, which will require dredging up to 40,000 cubic yards.

We plan to use a dredge called Pneuma pump, which is manufactured in Italy and represented in the United States by a Chicago firm. This type of dredge has never been used in Puget Sound country and it is a little experimental for us. The advantage of this particular dredge is that small amounts of water are used for the pipeline dredging; in other words, about 60 percent of the material that will enter the pipeline is sediments and about 40 percent water. Also, this dredge is excellent for controlling turbidity and has excellent control of dredging depth.

We plan on including predredging and postdredging monitoring sampling, which will be performed by EPA. There will be sampling during the actual dredging. We have looked at a number of disposal sites and methods and we have selected one. All of the areas have one thing in common and that is that no one wants the stuff.

The proposed disposal method is as follows: (1) We will pump, using the pneumadredge, into a watertight 4,000-yd^3 barge; (2) When full, the barge will be hauled 2 miles upstream to the disposal area; (3) The pneumadredge will then be placed into the barge and sediments will then be pumped into a disposal area.

The disposal area will be prepared beforehand and will be roughly 300 feet from the waterway. In the Seattle area, we have a tide drain from about plus 12 feet down to about minus 2 feet below MLLW. The disposal area bottom will be at 0 feet MLLW elevation. Consequently, this material will be placed in the water table. The disposal area will be filled to about the plus 14 foot level. The disposal area will be covered with 2-3 feet of clean dredged materials after dredging is completed and material has solidified. All effluents leaving the disposal area will be run through a sand filter to remove suspended sediments.

The cost of this operation will be between $300,000 and $500,000. This is about $2,500 per gallon of PCB's removed. This case study has been presented to you to show one removal operation and illustrate the problems involved in removal and disposal of hazardous materials. Thank you.

CHLORINATION OF WATERS FOR DISINFECTION—A STUDY OF THE PRODUCTION OF UNDESIRABLE CHLORINATED PRODUCTS

Richard Johnsen, Ph.D.*

The current emphasis on environmental preservation and human health is resulting in an increased use of chlorine for disinfection and waste treatment. Few efforts, if any, are being made by those proposing such procedures to determine the possible adverse impact of increased usage.

The use of chlorine for water treatment falls into two categories: protection of public health, and industrial use for antifouling and for waste treatment. In the United States, chlorination of municiple water supplies and wastewater treatment plant effluents is the most common procedure for disinfection. Most biologists who have maintained aquatic animals in laboratories are aware of the toxicity of chlorinated tap water. Brungs (ref. 1) thoroughly reviewed the current knowledge of the effects of residual chlorine on aquatic life. However, this paper is not concerned directly, per se, with chlorine toxicity but rather its reaction with other constituents of waste waters to produce unwanted chlorinated products. The utility of chlorine in water treatment is attributable to its toxicological characteristics and its oxidative capacity. Chlorine is employed to destroy pathogenic and nuisance bacteria and other microorganisms, to modify the chemical constituents of the water being treated (e.g., reduction of tastes and odors), or both.

In Fort Collins, as well as in most other Colorado cities, chlorination is used not only for disinfection of community water supplies, but also in the disinfection of effluent waters from the two sewage treatment plants. This latter water is released, usually directly, into the Cache La Poudre River, which is part of the South Platte River system.

There are two sewage treatment plants in Fort Collins, with the most recent one (onstream in 1969) currently undergoing a large expansion. The newer plant currently handles about 5.5 million gallons per day of waste waters with a near tripling in capacity nearing completion. Terminal chlorination at this plant site is carried out on the effluent stream from the clarifying tanks and just before the effluent enters the chlorination holding basin from which the water is released to the river.

The question that arises is whether this terminal chlorination treatment, with a desired disinfection as its goal, can give rise to undesired chlorinated products. Recently considerable concern was voiced in the national wire services over the findings by regulatory agencies of certain chlorinated compounds in the drinking waters of numerous communities (e.g., New Orleans) which are potentially dangerous to human health. Most of those cited to date include chlorinated alkanes (methanes and ethanes, etc.) and chlorinated phenols. The sources of these compounds were not ascertained but were being attributed to industrial effluents.

The high reactivity of chlorine with many organic compounds may provide another answer. Since sewage effluent waters from one community become the eventual drinking waters of communities downriver, it is of considerable importance that it be known that a disinfection process at one point does not contribute to the pollution burden at a second point. Another concern is the effect on aquatic fauna of such chlorinated compounds as may be formed by chlorination of waste waters. Although tremendous amounts of data are available on many chlorinated compounds having deleterious environmental effects (e.g., the insecticide DDT and the herbicide 2,4,5-T), very little is known about chlorinated compounds that may emanate from chlorinated sewage effluent waters.

Review of the Literature

A recent release of preliminary results of an Environmental Protection Agency (EPA) survey of U.S. drinking waters suggest that chemical contamination is a national problem (ref. 2). The report states that all 79 cities surveyed contained some amount of chloroform, ranging from 0.1 part per billion (ppb) to 311 ppb. This and other chemicals found were reported to be partially due to chlorination of drinking waters, Delfino (ref. 3), in response to earlier related reports, doubted the formation of chloroform in the chlorine disinfection process but called for more research in this area. Schwartz (ref. 4), in response to Delfino, cites the relative ease in which one may expect to find chlorinated compounds after chlorination considering all the possible organic compounds found just in sewage effluents. Laubusch (ref. 5) stated that when chlorine is added to water, a mixture of hypochlorous (NOCl) and hydrochloric (HCl) acids is formed and that this reaction is complete in a few seconds. He points out that under some conditions, chlor-addition or chlor-substitution products may be formed, but gives no further data. Gaffney (ref. 6), in a letter in *Science*, found polychlorinated biphenyls (PCB's) in a sewage treatment plant's trickling filter bed. These PCB's were the result of a high biphenyl influx from a textile mill coupled with waste water prechlorination.

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*Associate Professor, Pesticide Research Laboratory, Department of Zoology and Entomology, Colorado State University, Fort Collins, Colorado 80523.

Our interests in this area were rekindled with the paper by Carlson et al. (ref. 7). They reported that not only were 10 organic compounds, ranging from phenol to benzene, chlorinated but also that biphenyl, used as a fungicide, was chlorinated under various conditions to varying extents. This latter compound was of particular interest since we have been working with PCB's for several years as an industrial pollutant. Glaze et al. (ref. 8) found a number of chlorinated compounds by gas chromatography (GC) after extracting waste-water effluents containing from 10 to 100 ppm chlorine. Only chloroform was identified although it was evident from their chromatograms that higher molecular weight chlorinated compounds also were involved. Although levels of chlorine can be quite variable in effluent waters, depending on the chlorine metering devices and operator control, there are known to be some treatment plants that discharge effluents containing as high as 15 ppm total chlorine residual (ref. 9, p. 456).

The literature is replete with references to PCB's in aquatic animals, such as fish, crayfish, snails, shellfish, etc., in fish-eating birds, predatory birds, and other vertebrates, and the question that now arises is whether some of these PCB's are industrial pollutants or those produced by chlorination of waste waters.

Background Experience

We have studied and monitored PCB concentrations in digested sewage sludge, fish, and effluent waters for several years. We have shown that PCB levels have stabilized in sewage sludge at about 4-6 ppm, that they are in the effluent discharges from the sewage plants, and that they are concentrating in the fish in the Poudre River at levels often exceeding 5 ppm (refs. 10,11). Although we have tried, we have been unsuccessful in determining the source of PCB's into the sewage treatment plants. In addition, the gas chromatograms of extracts from effluent waters and fish tissues are not the same as those from digested sludge. If this is not due to some metabolic process, it may possibly be due to chlorination of the effluent water. This we would like to determine.

The fact that PCB's have been reported in effluents from paper mills which use biphenyl in slime control and chlorine in bleaching processes, gives credence to the preliminary work we report below and that already cited.

Preliminary Work

Recently we have conducted some preliminary work using biphenyl in water (5 ppm) to which was added chlorinated water to give chlorine concentrations of 8, 83, and 830 ppm. Under ambient conditions and after aging for 24 hours and 1 week, the samples were analyzed, along with suitable controls, by gas chromatography. The chromatograms shown in figure 1 indicate at least 8 peaks attributed to PCB's. If the individual chromatograms of A and C were superimposed over B, one would see striking overlaps although relative peak heights of most of the peaks aren't comparable. The standard (B) is Aroclor 1221, an industrial formulation containing 21 percent chlorine by weight. This is just one of eight formulations. It is evident that the higher level of chlorine resulted in higher chlorine substitution levels, as indicated by the longer retention times. The above data have not yet been quantitated nor have PCB isomer assignments been made to the various peaks. However, we have on hand over 50 pure PCB standards ranging from several monochloro to decachloro biphenyls which will be used for identification. Present work has indicated that biphenyl is chlorinated readily under conditions similar to those in the local sewage treatment plants. Other work done has involved use of sodium and calcium hypochlorite as chlorine sources. Discussion of these preliminary studies need not be included here since chlorine gas is used locally and we will confine our work to this source initially.

Work Plan

Since we have on hand a very sizable supply of chlorinated PCB isomers, it is our intent to study the chlorination process initially in the laboratory using biphenyl and later Aroclor 1221, to determine the parameters necessary for chlorination. Some of these parameters would be pH, concentration of reactants, reaction time and lastly, chlorine source. Since Fort Collins and most other communities use chlorine gas, and since that is what we have used primarily in the preliminary work, we will continue using this form. After this work has been completed, we plan on using effluent waters obtained from the sewage treatment plant and conduct time-course studies to determine extent of chlorination with time. Chlorine concentration would be the next factor studied. Probably of utmost concern is the unequivocal identification of the products formed. The fact that we have many of the potential products is extremely beneficial. The water samples will be routinely extracted with hexane and analyzed by GC using our Micotek MT-220 equipped with four columns and dual Ni 63 electron-capture detectors. An infrared spectrophotometer (Perkin-Elmer 337) is on hand for determination of

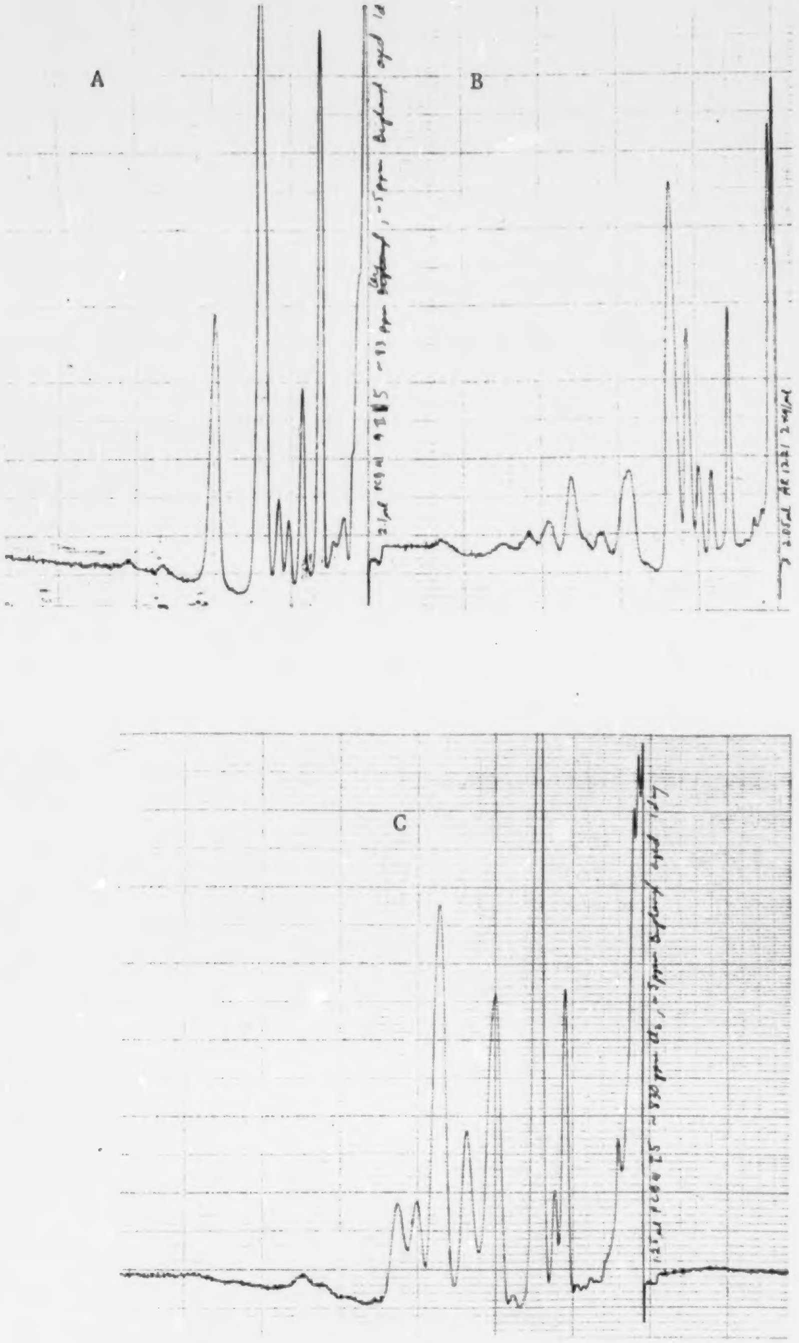

Figure 1. Gas chromatograms of extract of biphenyl reacted with Cl_2-water (83 ppm Cl) for 1 day (A), 830 ppm Cl for 1 day (C) and comparison to to Aroclor 1221 (B).

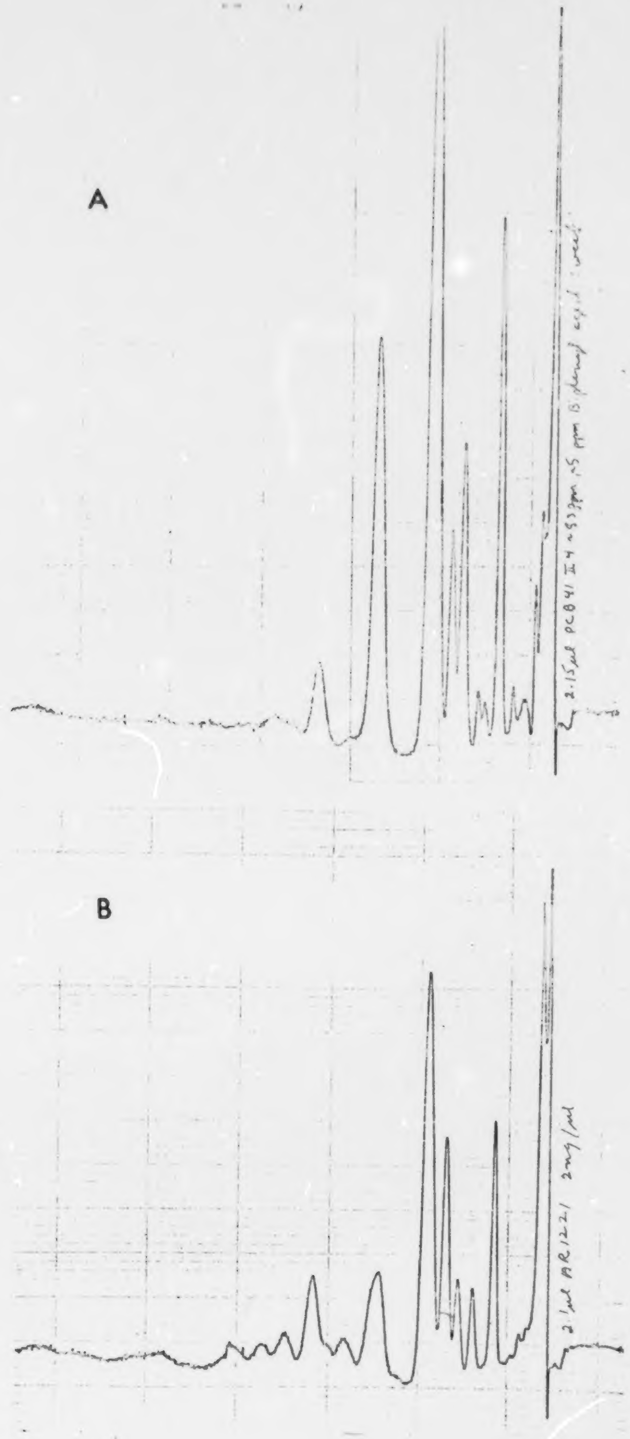

Figure 2. Gas chromatograms of extract of biphenyl reacted with Cl_2-water (83 ppm Cl) for 1 week (A) and comparison with Aroclor 1221 (B).

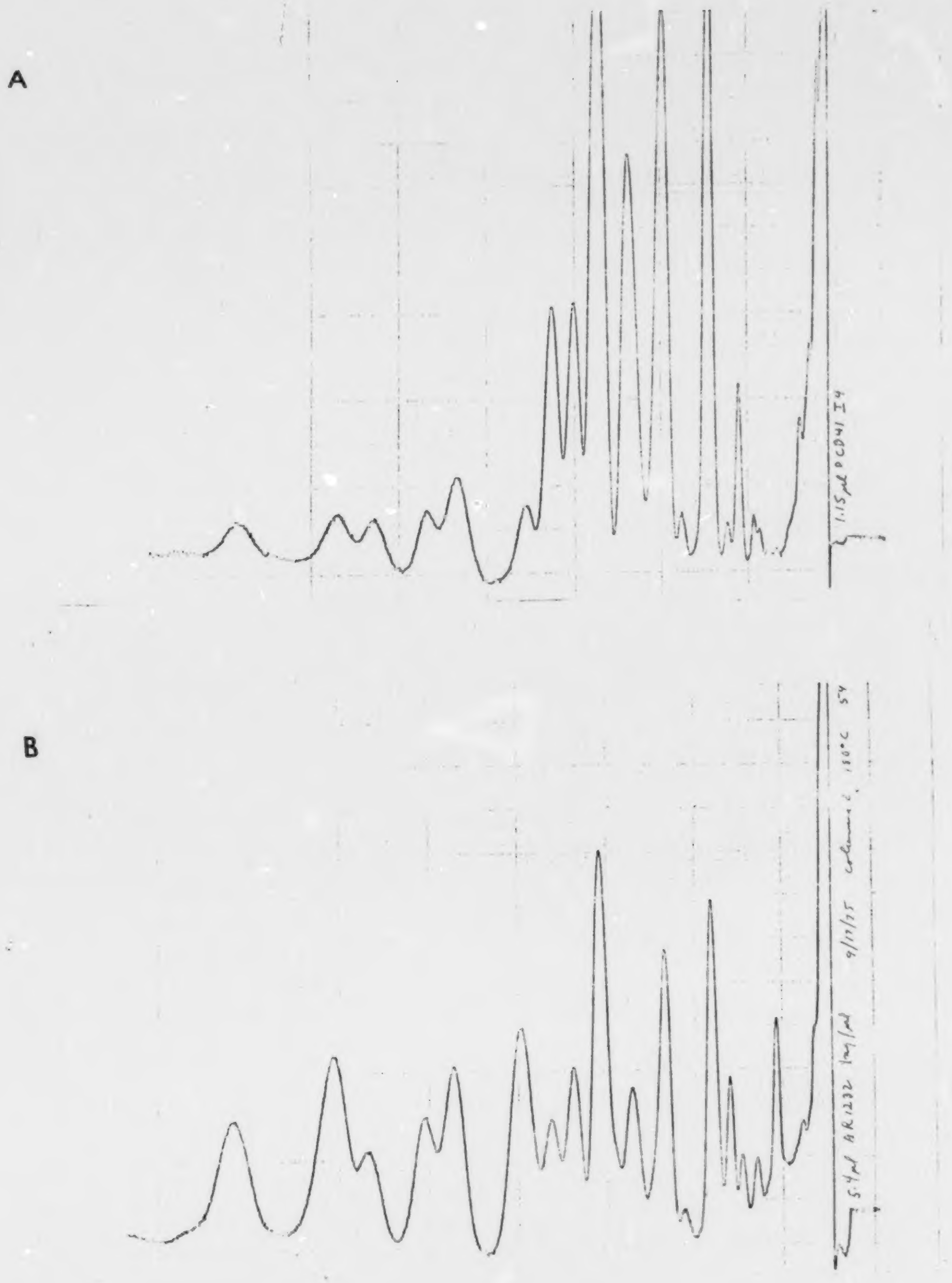

Figure 3. Gas chromatograms of extract of biphenyl reacted with Cl_2-water (830 ppm Cl) for 1 week (A) and comparison to Aroclor 1232 (B).

infrared spectra of products isolated by thin-layer chromatography. Confirmation of identities is planned using a mass spectrometer coupled with a GC.

Figure 2 illustrates the similarity of the extract (A) (the reaction of 83 ppm Cl_2 water with 5 ppm biphenyl incubated 1 week) with that of Aroclor 1221 but not in the same proportions. Using a light table, the peaks are superimposable on one another. This is true also of figure 3, where the chromatograms show the results of 830 ppm Cl and 5 ppm biphenyl incubated for 1 week in comparison to Aroclor 1232. It is evident that increasing chlorine dosages result in greater degrees of chlorination. The chromatograms in both figure 2 and figure 3 were very similar also after 1 day in incubation with the exception that the later eluting peaks are more pronounced. In figure 3 as in 2 above, all the peaks in the extract are in the Aroclor 1232. It was surprising to us that biphenyl was so readily chlorinated in light of its assumed unreactivity.

Further work is planned along these lines using shorter reaction time spans (hours), utilization of thiosulfate to terminate the reaction, use of CCl_4 or $CHCl_3$ instead of hexane as extracting solvent, and use of glass-distilled water (rather than from Barnstead still) distilled over basic $KMnO_4$. We have used reagent-grade Cl_2 gas and have not noticed any problem with bromine as an impurity. We would like to get access to a MS to check our peaks for chlorine content. We have checked the pH of the saturated Cl_2-water and found it to be 2.4 and after bubbling air through it, via a fritted disc, to be 2.45. So apparently the HOCl and HCl formed by the reaction of Cl_2 gas with water is quite stable. Some early peaks showing up in our blanks, especially the Cl_2-water blanks, are due possibly to the chlorination of hexane. This is the main reason we want to terminate the chlorination reaction with thiosulfate prior to extraction.

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* * * * * * * * * * * * * * * * * *

HERBERT GILNER (Tivian Laboratories, Providence, Rhode Island): I would like to address myself briefly to the methods used to gather information about polychlorinated biphenyls as environmental contaminants. I do not argue with the need, only with the methods.

It has been claimed both in the press and at this conference that of the 84 firms to whom letters were sent in August concerning the use of PCB's, only one has refused to respond. That company has been named in the press. That company is Tivian. However, to our knowledge, there is at least one other company which has also refused.

There are two distinct principles involved in this refusal. The first is that Tivian Laboratories does not use PCB's. On hearing this, the Environmental Protection Agency demanded information concerning Tivian's use of polychlorinated terphenyls, a series of compounds qualitatively different from PCB's. There has been, to our knowledge, no evidence that the PCT's accumulate in human tissues. Moreover, we state and have stated in the past that Tivian Laboratories do not pollute the environment.

Furthermore, and this gets us to the second principle, Tivian feels that the information demanded is proprietary and that EPA, having been set up by executive order, has no constitutional or statutory right to demand such information or to make threats concerning penalties for noncompliance. The company I represent feels that the harassing activities of the EPA, including trial by the press, are totally illegal.

EPA has sought to vilify the name of Tivian by passing its name to the press, claiming that Tivian has contaminated the environment with PCB's, which our company does not use.

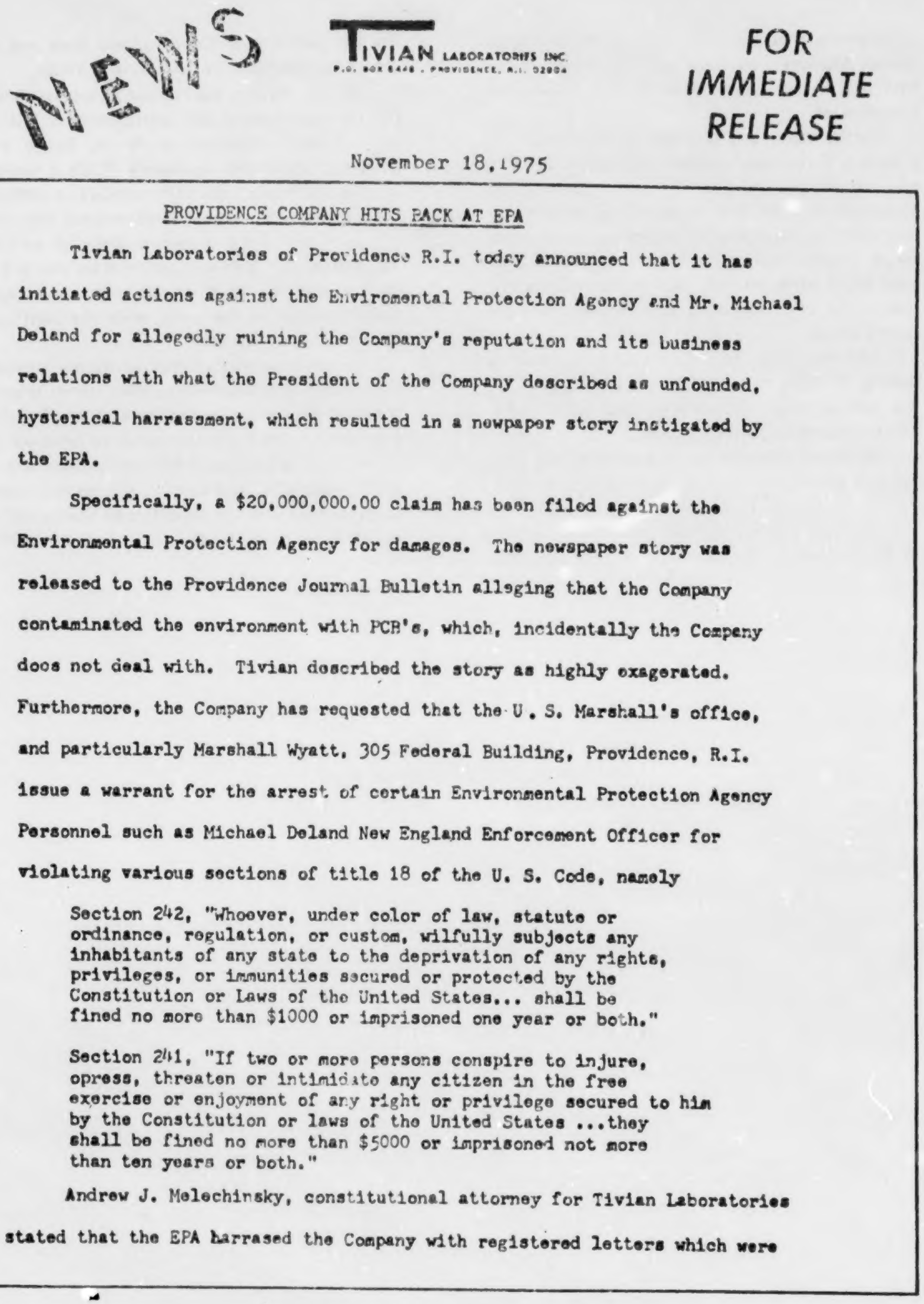
Tivian Laboratories has filed a $20 million damage suit against EPA for this release of false information to the public. Tivian has further requested the U.S. Marshall's Office in Providence, Rhode Island, to issue a warrant for the arrest of EPA personnel for the violation of Constitutional laws and congressional statutes in its dealings with Tivian.

It is ironic that the company most responsible for the manufacture and distribution of PCB's in this country continues to do so, but a small company which does not handle PCB's is harassed.

At our request, the EPA supplied us with a list of companies to whom questionnaires were sent. However, the names of certain firms, as we heard again yesterday, were excized from the list. Is it not strange that the name of one company is pejoratively released to the press while the identity of others is concealed? Why?

Is it not capricious that under our unelected administration in Washington, these names as well as those of the companies dealing with the Arabs and cooperating with the Arab boycott of Israel are kept secret? The objectives of EPA are without a doubt useful, necessary, and worthy, and we still support them, but we will not submit to an agency which is so blatantly being used as a tool of an unethical policy.

Figure 1, literature handed out by Tivian Laboratories, is on the following pages.



NEWS

TIVIAN LABORATORIES INC.
P.O. BOX 6448 . PROVIDENCE, R.I. 02904

FOR
IMMEDIATE
RELEASE

November 18, 1975

PROVIDENCE COMPANY HITS BACK AT EPA

Tivian Laboratories of Providence R.I. today announced that it has initiated actions against the Enviromental Protection Agency and Mr. Michael Deland for allegedly ruining the Company's reputation and its business relations with what the President of the Company described as unfounded, hysterical harrassment, which resulted in a newpaper story instigated by the EPA.

Specifically, a $20,000,000.00 claim has been filed against the Environmental Protection Agency for damages. The newspaper story was released to the Providence Journal Bulletin alleging that the Company contaminated the environment with PCB's, which, incidentally the Company does not deal with. Tivian described the story as highly exagerated. Furthermore, the Company has requested that the U. S. Marshall's office, and particularly Marshall Wyatt, 305 Federal Building, Providence, R.I. issue a warrant for the arrest of certain Environmental Protection Agency Personnel such as Michael Deland New England Enforcement Officer for violating various sections of title 18 of the U. S. Code, namely

> Section 242, "Whoever, under color of law, statute or ordinance, regulation, or custom, wilfully subjects any inhabitants of any state to the deprivation of any rights, privileges, or immunities secured or protected by the Constitution or Laws of the United States... shall be fined no more than $1000 or imprisoned one year or both."
>
> Section 241, "If two or more persons conspire to injure, opress, threaten or intimidate any citizen in the free exercise or enjoyment of any right or privilege secured to him by the Constitution or laws of the United States ...they shall be fined no more than $5000 or imprisoned not more than ten years or both."

Andrew J. Melechinsky, constitutional attorney for Tivian Laboratories stated that the EPA harrased the Company with registered letters which were

Figure 1. Tivian Laboratories handout.

refused by Tivian. When Tivian requested the exact contents of these letters and their nature, they were not given a definite answer as to their content. After Tivian had refused these letters, a letter was served upon Tivian by a U.S. Marshall, which contained a very lengthy questionaire form.

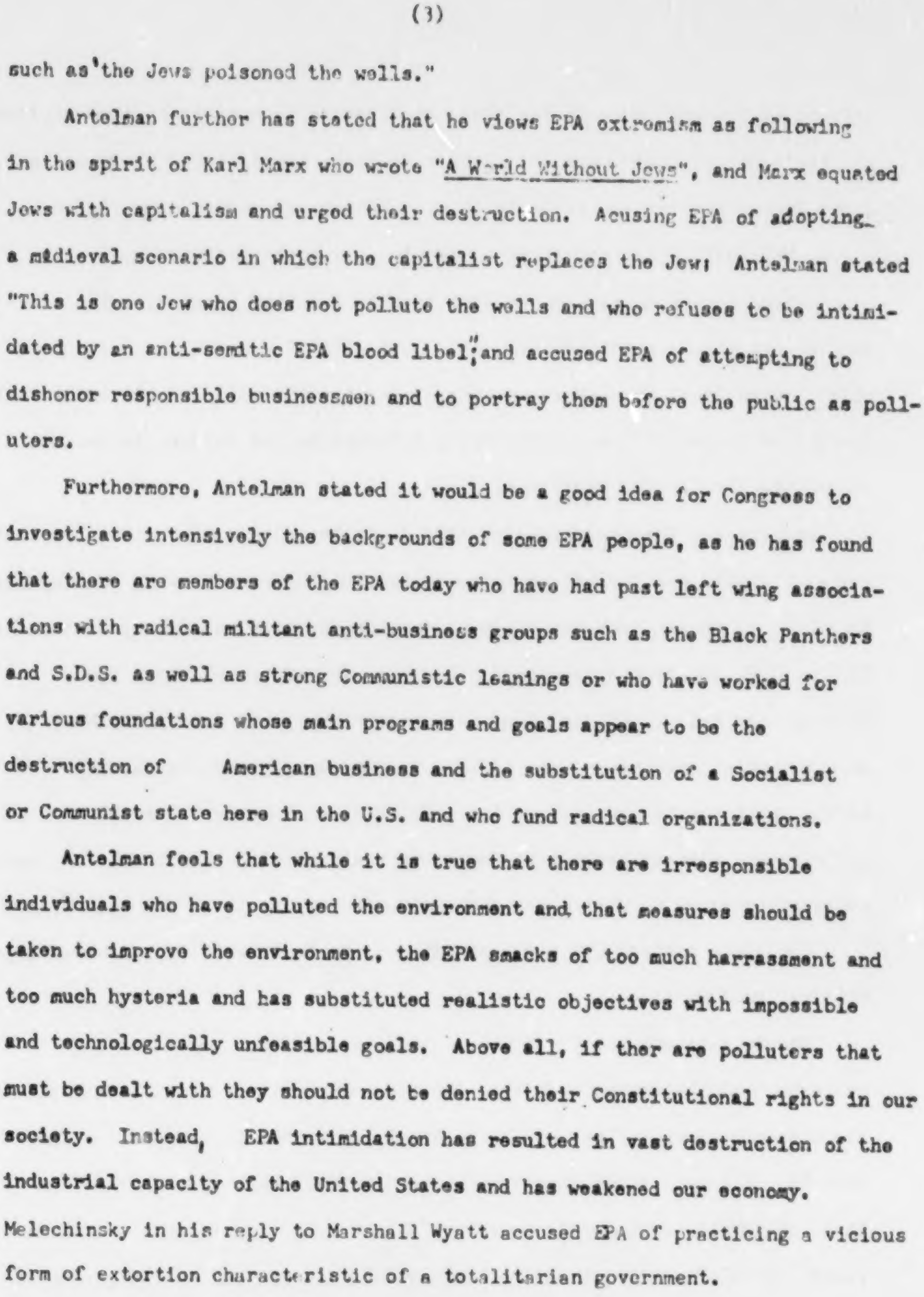
Melechinsky charges the EPA with several Constitutional violations in sending out the form particularly Amendment XIII of the U.S. Constitution, which states "Neither slavery nor involuntary servitude.....shall exist within the United States..." Any involuntary response to the subject letter would constitute involuntary servitude.

Melechinsky further maintains that it is becoming a disgusting practice for many states and federal agencies to harrass businessmen with lengthy forms. He asks: 1. Do not such forms add to a tax bill which is already fantastically high? 2. Who pays companies for the time and effort required for filling them out? 3. If such funds are available, isn't that a further unwarranted expenditure of tax monies? 4. How can governments justify adding another nuisance load to the many which various governmental agencies are already seeking to impose on businesses? 5. Isn't this an invasion of privacy? 6. What Constitutional authority is there for such forms?

The claim against EPA has been filed by Tivian's Vice-President of Legal Affairs, Paul Wallins.

Marvin Antelman the President of the Corporation, has stated that he believes that the Agency harrassed him because of his constant criticism, in his capacity as an officer of the National Leadership Conference for The Security of Israel of Kissinger and Ford for their hard line against the State of Israel and Commerce Secretary's Mortton's support of the Arab Boycott. Antelman stated that the Company will attempt in its litigation to document this. In a terse reply to Michael Deland Antelman stated "This most recent incident smacks very much like that of a medieval antisemitic libel

Figure 1. (con.).



(3)

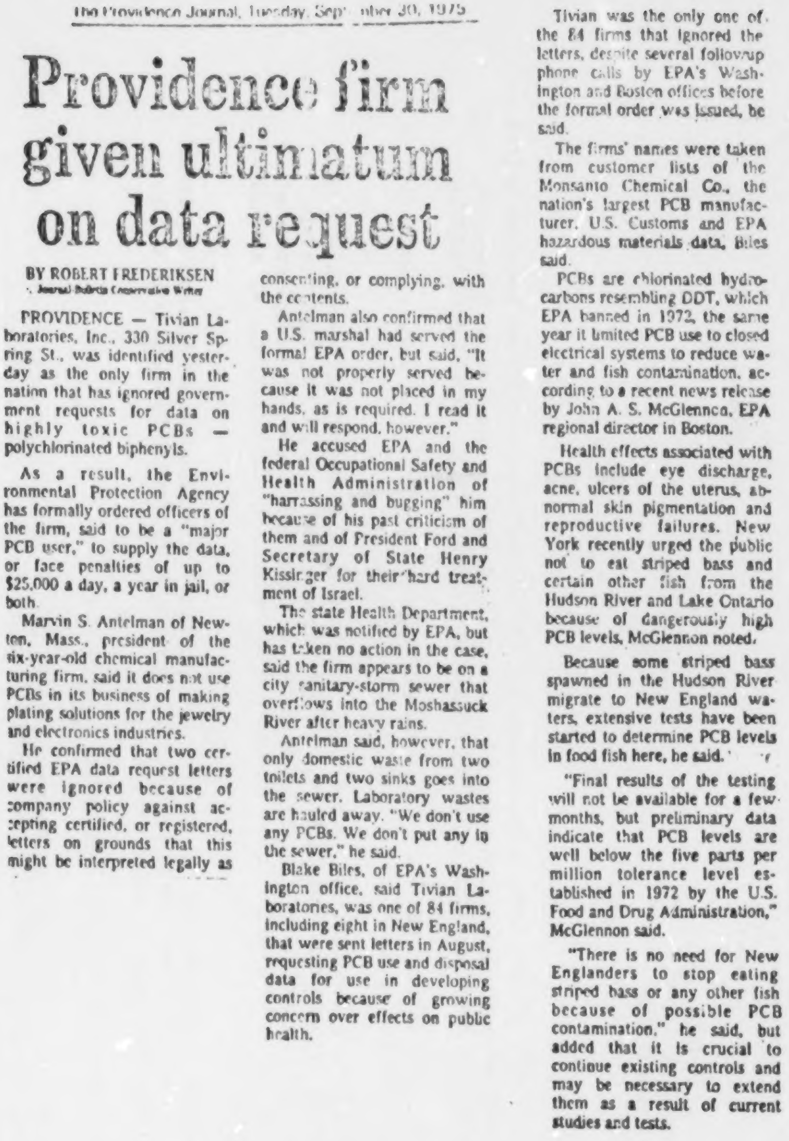
such as 'the Jews poisoned the wells."

Antelman further has stated that he views EPA extremism as following in the spirit of Karl Marx who wrote "A World Without Jews", and Marx equated Jews with capitalism and urged their destruction. Acusing EPA of adopting a medieval scenario in which the capitalist replaces the Jew; Antelman stated "This is one Jew who does not pollute the wells and who refuses to be intimidated by an anti-semitic EPA blood libel"; and accused EPA of attempting to dishonor responsible businessmen and to portray them before the public as polluters.

Furthermore, Antelman stated it would be a good idea for Congress to investigate intensively the backgrounds of some EPA people, as he has found that there are members of the EPA today who have had past left wing associations with radical militant anti-business groups such as the Black Panthers and S.D.S. as well as strong Communistic leanings or who have worked for various foundations whose main programs and goals appear to be the destruction of American business and the substitution of a Socialist or Communist state here in the U.S. and who fund radical organizations.

Antelman feels that while it is true that there are irresponsible individuals who have polluted the environment and that measures should be taken to improve the environment, the EPA smacks of too much harrassment and too much hysteria and has substituted realistic objectives with impossible and technologically unfeasible goals. Above all, if ther are polluters that must be dealt with they should not be denied their Constitutional rights in our society. Instead, EPA intimidation has resulted in vast destruction of the industrial capacity of the United States and has weakened our economy. Melechinsky in his reply to Marshall Wyatt accused EPA of practicing a vicious form of extortion characteristic of a totalitarian government.

Figure 1. (con.).



The Providence Journal, Tuesday, September 30, 1975

Providence firm given ultimatum on data request

BY ROBERT FREDERIKSEN
Journal-Bulletin Conservation Writer

PROVIDENCE — Tivian Laboratories, Inc., 330 Silver Spring St., was identified yesterday as the only firm in the nation that has ignored government requests for data on highly toxic PCBs — polychlorinated biphenyls.

As a result, the Environmental Protection Agency has formally ordered officers of the firm, said to be a "major PCB user," to supply the data, or face penalties of up to $25,000 a day, a year in jail, or both.

Marvin S. Antelman of Newton, Mass., president of the six-year-old chemical manufacturing firm, said it does not use PCBs in its business of making plating solutions for the jewelry and electronics industries.

He confirmed that two certified EPA data request letters were ignored because of company policy against accepting certified, or registered, letters on grounds that this might be interpreted legally as consenting, or complying, with the contents.

Antelman also confirmed that a U.S. marshal had served the formal EPA order, but said, "It was not properly served because it was not placed in my hands, as is required. I read it and will respond, however."

He accused EPA and the federal Occupational Safety and Health Administration of "harrassing and bugging" him because of his past criticism of them and of President Ford and Secretary of State Henry Kissinger for their hard treatment of Israel.

The state Health Department, which was notified by EPA, but has taken no action in the case, said the firm appears to be on a city sanitary-storm sewer that overflows into the Moshassuck River after heavy rains.

Antelman said, however, that only domestic waste from two toilets and two sinks goes into the sewer. Laboratory wastes are hauled away. "We don't use any PCBs. We don't put any in the sewer," he said.

Blake Biles, of EPA's Washington office, said Tivian Laboratories, was one of 84 firms, including eight in New England, that were sent letters in August, requesting PCB use and disposal data for use in developing controls because of growing concern over effects on public health.

Tivian was the only one of the 84 firms that ignored the letters, despite several followup phone calls by EPA's Washington and Boston offices before the formal order was issued, he said.

The firms' names were taken from customer lists of the Monsanto Chemical Co., the nation's largest PCB manufacturer, U.S. Customs and EPA hazardous materials data, Biles said.

PCBs are chlorinated hydrocarbons resembling DDT, which EPA banned in 1972, the same year it limited PCB use to closed electrical systems to reduce water and fish contamination, according to a recent news release by John A. S. McGlennon, EPA regional director in Boston.

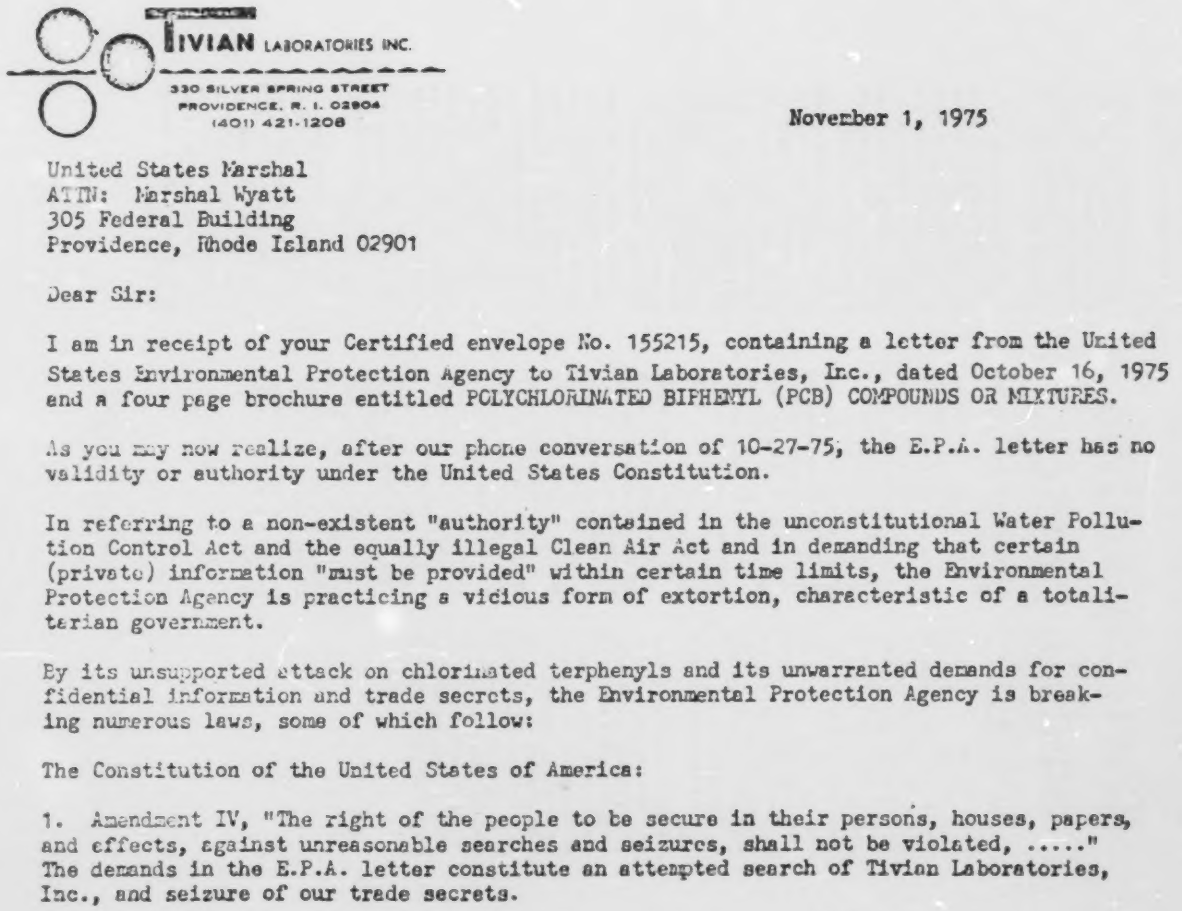
Health effects associated with PCBs include eye discharge, acne, ulcers of the uterus, abnormal skin pigmentation and reproductive failures. New York recently urged the public not to eat striped bass and certain other fish from the Hudson River and Lake Ontario because of dangerously high PCB levels, McGlennon noted.

Because some striped bass spawned in the Hudson River migrate to New England waters, extensive tests have been started to determine PCB levels in food fish here, he said.

"Final results of the testing will not be available for a few months, but preliminary data indicate that PCB levels are well below the five parts per million tolerance level established in 1972 by the U.S. Food and Drug Administration," McGlennon said.

"There is no need for New Englanders to stop eating striped bass or any other fish because of possible PCB contamination," he said, but added that it is crucial to continue existing controls and may be necessary to extend them as a result of current studies and tests.

Figure 1. (con.).



TIVIAN LABORATORIES INC.
330 SILVER SPRING STREET
PROVIDENCE, R. I. 02904
(401) 421-1208

November 1, 1975

United States Marshal
ATTN: Marshal Wyatt
305 Federal Building
Providence, Rhode Island 02901

Dear Sir:

I am in receipt of your Certified envelope No. 155215, containing a letter from the United States Environmental Protection Agency to Tivian Laboratories, Inc., dated October 16, 1975 and a four page brochure entitled POLYCHLORINATED BIPHENYL (PCB) COMPOUNDS OR MIXTURES.

As you may now realize, after our phone conversation of 10-27-75, the E.P.A. letter has no validity or authority under the United States Constitution.

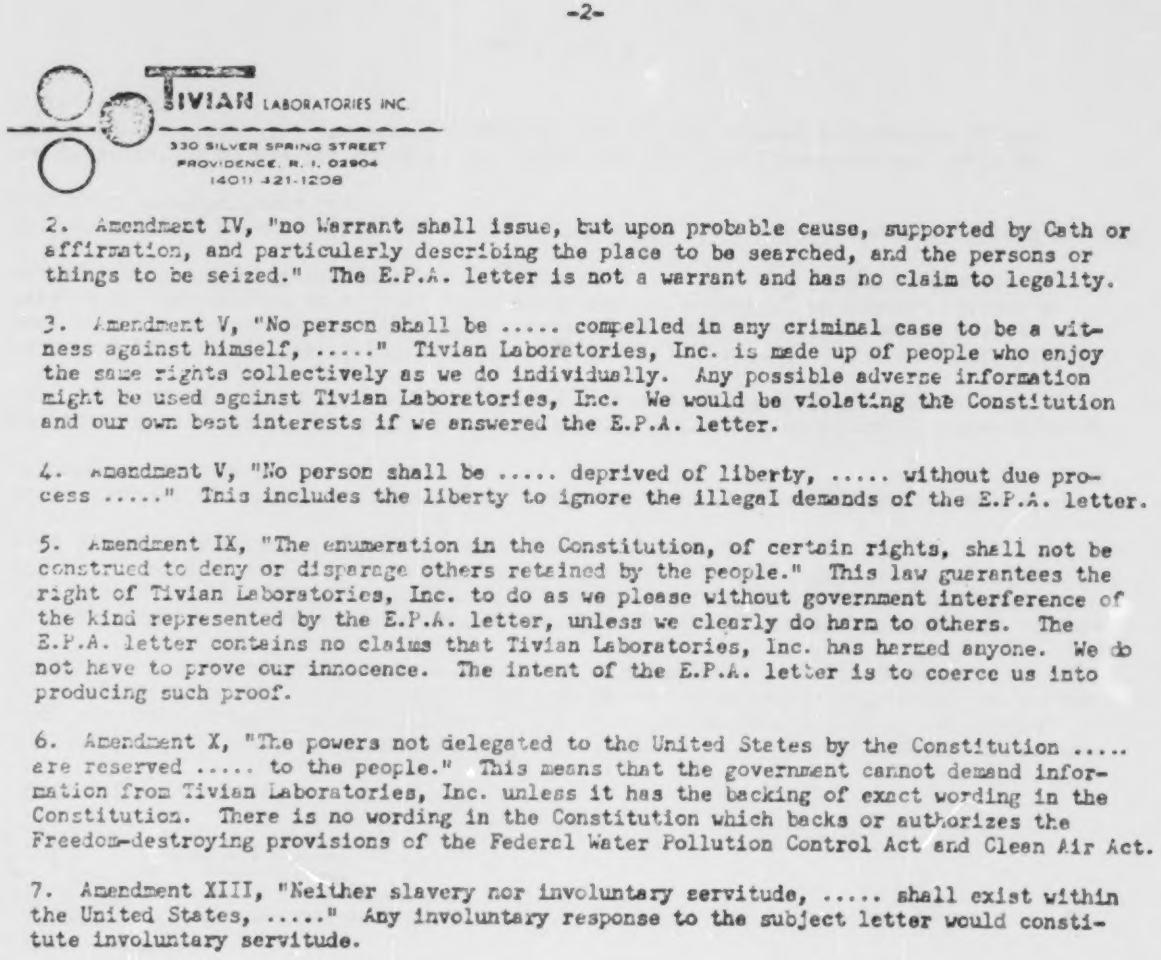
In referring to a non-existent "authority" contained in the unconstitutional Water Pollution Control Act and the equally illegal Clean Air Act and in demanding that certain (private) information "must be provided" within certain time limits, the Environmental Protection Agency is practicing a vicious form of extortion, characteristic of a totalitarian government.

By its unsupported attack on chlorinated terphenyls and its unwarranted demands for confidential information and trade secrets, the Environmental Protection Agency is breaking numerous laws, some of which follow:

The Constitution of the United States of America:

1. Amendment IV, "The right of the people to be secure in their persons, houses, papers, and effects, against unreasonable searches and seizures, shall not be violated, ..." The demands in the E.P.A. letter constitute an attempted search of Tivian Laboratories, Inc., and seizure of our trade secrets.

Figure 1. (con.).



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TIVIAN LABORATORIES INC.
330 SILVER SPRING STREET
PROVIDENCE, R. I. 02904
(401) 421-1208

2. Amendment IV, "no Warrant shall issue, but upon probable cause, supported by Oath or affirmation, and particularly describing the place to be searched, and the persons or things to be seized." The E.P.A. letter is not a warrant and has no claim to legality.

3. Amendment V, "No person shall be ..... compelled in any criminal case to be a witness against himself, ..." Tivian Laboratories, Inc. is made up of people who enjoy the same rights collectively as we do individually. Any possible adverse information might be used against Tivian Laboratories, Inc. We would be violating the Constitution and our own best interests if we answered the E.P.A. letter.

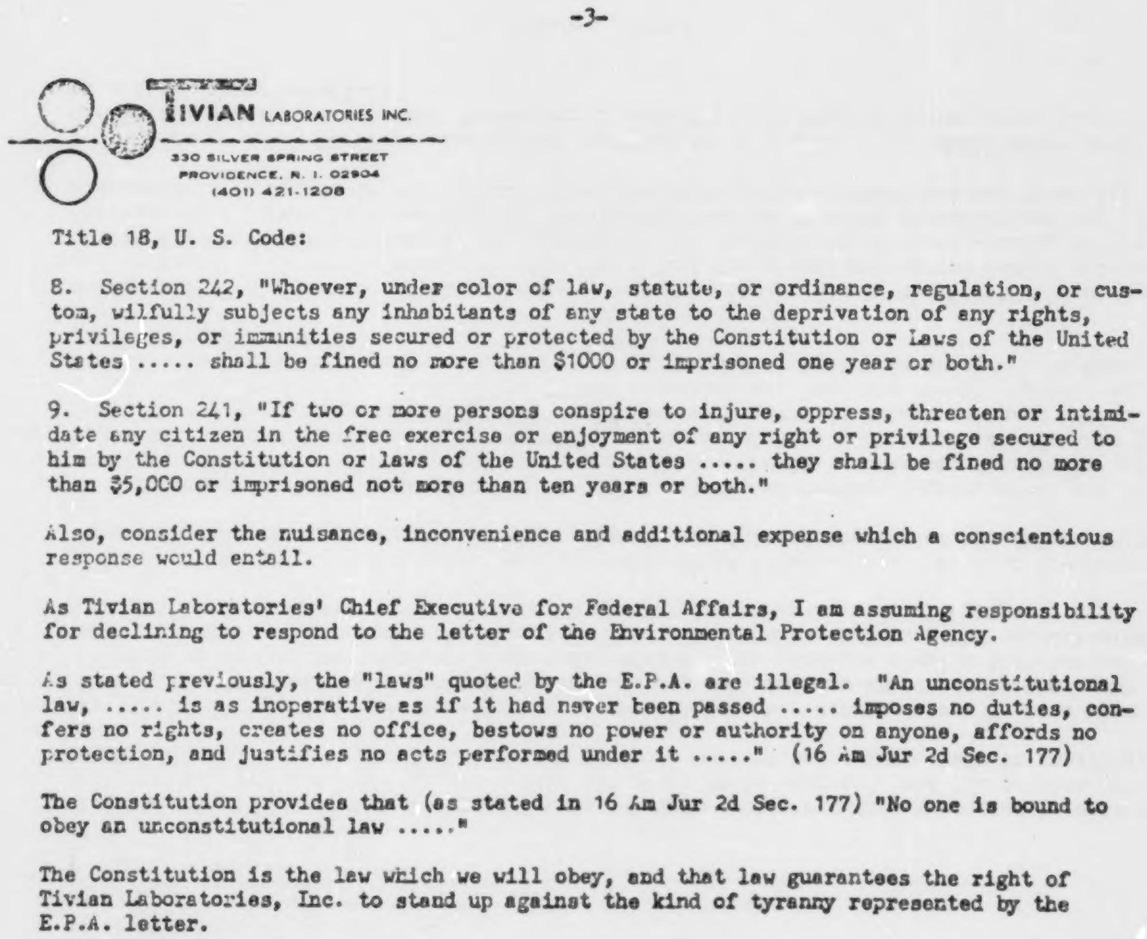
4. Amendment V, "No person shall be ..... deprived of liberty, ... without due process ....." This includes the liberty to ignore the illegal demands of the E.P.A. letter.

5. Amendment IX, "The enumeration in the Constitution, of certain rights, shall not be construed to deny or disparage others retained by the people." This law guarantees the right of Tivian Laboratories, Inc. to do as we please without government interference of the kind represented by the E.P.A. letter, unless we clearly do harm to others. The E.P.A. letter contains no claims that Tivian Laboratories, Inc. has harmed anyone. We do not have to prove our innocence. The intent of the E.P.A. letter is to coerce us into producing such proof.

6. Amendment X, "The powers not delegated to the United States by the Constitution ..... are reserved ..... to the people." This means that the government cannot demand information from Tivian Laboratories, Inc. unless it has the backing of exact wording in the Constitution. There is no wording in the Constitution which backs or authorizes the Freedom-destroying provisions of the Federal Water Pollution Control Act and Clean Air Act.

7. Amendment XIII, "Neither slavery nor involuntary servitude, ... shall exist within the United States, ..." Any involuntary response to the subject letter would constitute involuntary servitude.

Figure 1. (con.).



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TIVIAN LABORATORIES INC.
330 SILVER SPRING STREET
PROVIDENCE, R. I. 02904
(401) 421-1208

Title 18, U. S. Code:

8. Section 242, "Whoever, under color of law, statute, or ordinance, regulation, or custom, wilfully subjects any inhabitants of any state to the deprivation of any rights, privileges, or immunities secured or protected by the Constitution or Laws of the United States ..... shall be fined no more than $1000 or imprisoned one year or both."

9. Section 241, "If two or more persons conspire to injure, oppress, threaten or intimidate any citizen in the free exercise or enjoyment of any right or privilege secured to him by the Constitution or laws of the United States ..... they shall be fined no more than $5,000 or imprisoned not more than ten years or both."

Also, consider the nuisance, inconvenience and additional expense which a conscientious response would entail.

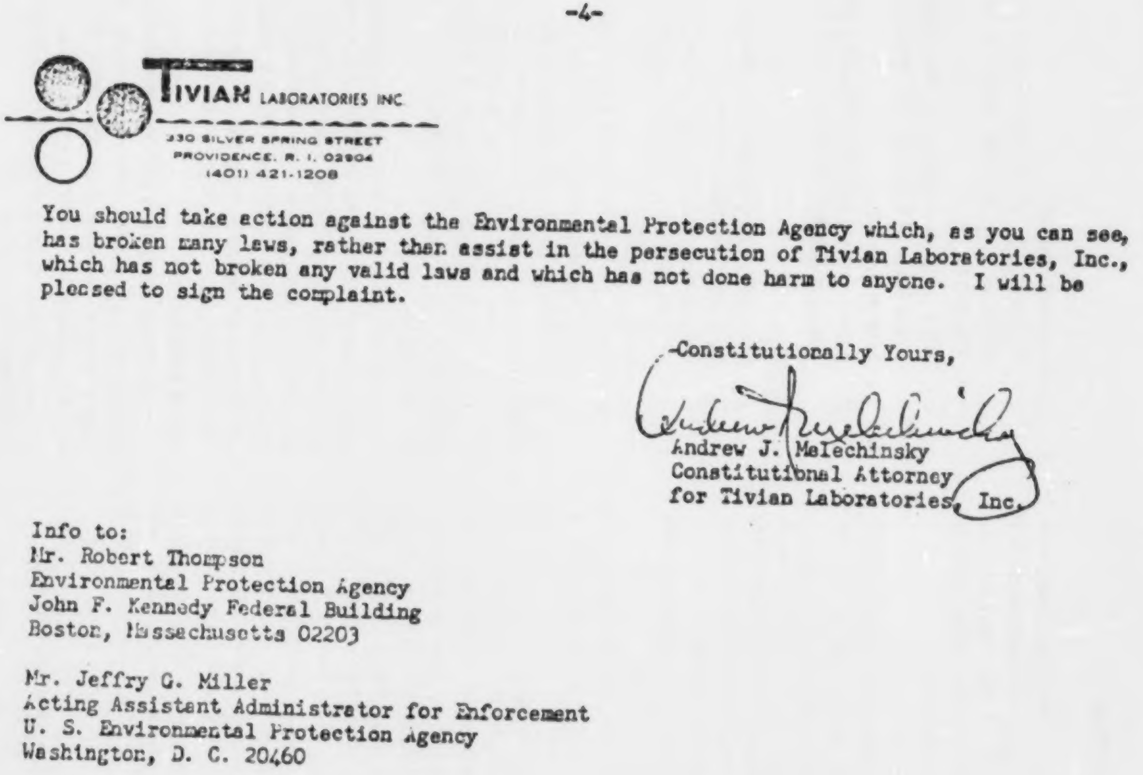
As Tivian Laboratories' Chief Executive for Federal Affairs, I am assuming responsibility for declining to respond to the letter of the Environmental Protection Agency.

As stated previously, the "laws" quoted by the E.P.A. are illegal. "An unconstitutional law, ... is as inoperative as if it had never been passed ..... imposes no duties, confers no rights, creates no office, bestows no power or authority on anyone, affords no protection, and justifies no acts performed under it ....." (16 Am Jur 2d Sec. 177)

The Constitution provides that (as stated in 16 Am Jur 2d Sec. 177) "No one is bound to obey an unconstitutional law ....."

The Constitution is the law which we will obey, and that law guarantees the right of Tivian Laboratories, Inc. to stand up against the kind of tyranny represented by the E.P.A. letter.

Figure 1. (con.).



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TIVIAN LABORATORIES INC

330 SILVER SPRING STREET
PROVIDENCE, R. I. 02904
(401) 421-1208

You should take action against the Environmental Protection Agency which, as you can see, has broken many laws, rather than assist in the persecution of Tivian Laboratories, Inc., which has not broken any valid laws and which has not done harm to anyone. I will be pleased to sign the complaint.

Constitutionally Yours,

Andrew J. Melechinsky
Constitutional Attorney
for Tivian Laboratories, Inc.

Info to:
Mr. Robert Thompson
Environmental Protection Agency
John F. Kennedy Federal Building
Boston, Massachusetts 02203

Mr. Jeffry G. Miller
Acting Assistant Administrator for Enforcement
U. S. Environmental Protection Agency
Washington, D. C. 20460

Figure 1. (con.).

THE NEED FOR COST-BENEFIT ANALYSIS IN TOXIC SUBSTANCE USAGE

A. Eatock*

INTRODUCTION

Yesterday, mercury had the limelight; today, it is PCB's; tomorrow—what? We face a recurrent problem in dealing with toxic substances in the national-social context. Thus, this paper discusses the overall problem of toxic substances rather than just PCB's.

The purpose of this paper is threefold:

a. to show the absolute necessity of cost-benefit analysis in achieving an equitable balance between private and social costs and benefits;
b. to illustrate the need in cost-benefit analysis for researchers to be specific in delineating the chain of consequences together with the risks involved in toxic substances; and
c. to illustrate the elements of a simplified cost-benefit analysis based on PCB's.

Conceptually, cost-benefit analysis is quite simple in that one places a value on the costs of an action or program and a value on the benefits resulting therefrom. If the dollar benefits exceed the dollar cost, then one proceeds with the action or program. However, simple ideas do tend to get complicated in practice and cost-benefit analysis is no exception. One complication, which will be ignored hereafter, is what interest rate to use in finding the present value of future benefits such that they can be compared to the costs. Another complication is how to value intangibles (such as good health and aesthetics) that do not have an established price from being traded on the open market. There is no easy, or even commonly accepted, method for valuing the unintended effects of toxic substances resulting in social costs. This results in considerable conflict due to the judgmental valuing in estimating social costs and, frequently, the ignoring of the whole issue of intangibles.

MAGNITUDE OF THE PCB PROBLEM

To illustrate one aspect of the magnitude of the PCB problem and to lay the groundwork for a cost-benefit analysis, a hypothetical case on the accumulated PCB loading to the Lower Great Lakes from U.S. sources is worked out. There are three categories of inputs as follows:

1. Uncontrolled Imports:
   a. Estimate (ref. 1) of uncontrolled imports of PCB's as part of other products in 1972 ≅ 375,000 lb.
   b. 1971 U.S. population in the Lower Great Lakes Basin (ref. 2) $13 \times 10^6 \cong 6.3$ percent of total population.
   c. Lower Great Lakes water volume converted to weight ≅ $4{,}575 \times 10^{12}$ lb.

Assumptions:

a. The PCB distribution is similar to the population distribution (ref. 2).
b. The continuing flow of import products results in a continuing equivalent amount going into the environment.
c. Thirty percent of PCB's are of the persistent (nonbiodegradable) types (ref. 4).

Then, 375,000 lb of uncontrolled imports result in an equivalent lake water PCB load of 1.5 ppt. Please note the word "equivalent;" in actual fact, the lake water has a very much lower level since PCB's tend to accumulate in the biota and sediment.

2. Existing Pool:

It is reported (ref. 3) that the U.S. consumption was approximately 1 billion lb of PCB's during the 40-year period from 1930 to 1970. The majority of this PCB consumption must now be in the environment, primarily in garbage dumps and sediment. If 0.1 percent of this pool is released annually from the various sources, then the equivalent lake load is 4 ppt PCB's in the water.

3. Continuing Use:

The third category is continuing authorized use for transformers and power factor capacitors, which constitute a consumption of more than 50×10^6 lb/yr (ref. 4). An assumption of a 0.1 percent leakage rate to the environment results in an equivalent lake load of 0.2 ppt in the water.

This is the U.S. loading on the Lower Great Lakes, to which the Canadian contribution must be added. Keeping in mind that these figures are based on unsupported assumptions, it is still interesting to compare them with the proposed water quality target of 1 ppt. It does indicate that, since PCB's are so persistent, the problem will have to be lived with for a long time. How far to go in rectifying the problem is where

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*Social Sciences Division, Inland Waters Directorate-Ontario Region, Burlington, Ontario, Canada L7R 4A6.

A COST-BENEFIT FORMAT

Benefits

The chief benefit of PCB's is their nonflammability in the liquid phase. This has resulted in *only* PCB-cooled transformers and power factor capacitors being allowed in the upper floors of tall buildings. On similar safety grounds, it is the only fluid permissible for use in electrostatic precipitators that keep the particulate pollution from going up the stack of most industries. No estimates for the value of these benefits, and others which are not considered, are readily available, but they must be substantial or the OECD (ref. 8) group would not have recommended continuing these uses. While not ignoring the necessity of confirming these benefits, the cost-benefit analysis may be simplified to the tradeoffs between direct costs of controlled usage and the cost of damage to the natural and human environment.

At this point, the three sources of contamination can be incorporated in the cost-benefit analysis, as shown in table 1. These figures give some indication of the most cost-effective tradeoffs between various control methods and damages to the natural and human environment.

COST OF CONTROLLING USAGE

The cost of controlling the authorized usages of PCB's consists of the direct and indirect costs (table 2). The direct costs include containment devices to avoid leakages and losses; the handling of contaminated containers, cloths, etc.; the decontamination of equipment; the incineration of contaminated cleaning solvents, clothes, etc.; and the cost of monitoring and policing these activities to minimize leakages to the environment.

Indirect costs could include the monitoring and policing required to reduce unauthorized imports of PCB's as part of other products, and sewage incineration to reduce the environmental levels of PCB's. This latter indirect cost is of considerable interest since high-temperature incineration appears to be the only practical method of destroying PCB's, and sewage sludge incineration has the potential to break the continuing cycle of PCB through the environment. In Ontario, approximately 40 percent of the sewage sludge is incinerated, principally by the larger centers that are the main sources of PCB's. Unfortunately, temperatures well in excess of 860° C are required to destroy PCB's and the normal incinerator operation is just at or below this temperature. Further, since considerable energy is required to burn the wet sludge, optimization of sewage plant operations dictates operating at even lower operating temperatures as the cost of fuel increases. Obviously, there is a need to optimize sewage plant incinerator operations with the destruction of PCB's included in the criteria. The higher cost of fuel can possibly be alleviated by exhaust gas heat recovery to dry the incoming sludge, which apparently is not common practice at present.

SOCIAL COSTS

Social costs, including damage to the environment, as shown in table 3, are again split into direct and indirect costs. The direct costs include loss of commercial fishing and loss of recreational industry business due to the contamination by PCB's of the larger predatory fish which are at the top of the bioaccumulation chain (ref. 5). Another direct cost is the loss of foodstuffs due to contamination during production and packaging. Should PCB's continue to be dispersed to the environment from sewage incineration stacks, and sewage sludge utilized as fertilizer, there is a good possibility that the level of PCB's in milk, presently as high as 0.1 ppm near industrial centers in southern Ontario, will continue to rise and result in value lost when the milk is not fit for consumption. The occasional poisoning of industrial workers working with PCB's, or the poisoning of individuals in the general population due to accidental spills, gives rise to costs from lost work time and medical attention.

Indirect costs (table 4) include such things as fish-eating birds that have all but disappeared from the lower lakes; these include osprey, bald eagle, cormorant, bittern, herons, and loons (ref. 6). The herring gulls would also disappear except for the influx from the East Coast and the Upper Great Lakes (refs. 6,7). There are two aspects related to the loss of these birds. One is that, if the predator fish population declines, the alewife population will increase, and if the gull population declines, who is going to clean up, and at what cost, the mess resulting from massive alewife dieoffs such as we had a few years ago. This is what can happen if the balance of nature is disturbed. However, the main interest at the moment is the social cost aspect. How does one cost the aesthetic value of the bird population? This problem is typical of the multitude of social costs that must be considered, and there is no easy answer or agreement on methods.

EXAMPLE OF SOCIAL COST ANALYSIS

One approach taken to illustrate the problem is as follows. According to the Federation of Ontario Naturalists, there are approximately 750 bird watchers

Table 1. Functional organizational model

Benefits	Due existing pool	Due continuing usage	Due uncontrolled imports
Transformer and power factor capacitators safety			
Reduction in property loss			
Reduction in loss of life			
Reduction in mdeical costs			
Reduction in employment loss			
Reduction in pollution (electrostatic precipitators)			
Reduction in cost of controlling pollution			

Table 2. Costs of controlling the authorized usages of PCB's

	Risk (cost) due existing pool	Risk (cost) due continuing usage	Risk (cost) due uncontrolled imports
Cost of controlling usage			
Direct costs			
Containment			
Contaminated articles disposal			
Decontamination			
Incineration			
Monitoring and policing			
Indirect costs			
Monitoring and policing (imports)			
Sewage sludge incineration			

Table 3. Social costs of PCB's

	Risk (cost) due existing pool	Risk (cost) due continuing usage	Risk (cost) due uncontrolled imports
Cost of controlling usage			
Social costs (environmental damage costs)			
Direct costs			
Commercial fisheries			
Recreation industry			
Contaminated food			
Worker lost time			
Worker medical costs			
Nonrecycling of paper			

in Ontario who report on migratory birds and spend approximately 500 hours a year observing; there are another 15,000 bird watchers who pay $12.00 per year to belong to the organization, and it may be arbitrarily assumed that they spend 10 days a year watching birds, for a total of 60 hr/yr. Assuming this is an exponential function tailing off to the 8 millionth person in Ontario, who never looks at birds, it can be calculated that 125,000 hours are spent in bird watching. Again being arbitrary, it could be assumed that 10 percent of the pleasure in bird watching for Ontario residents is lost with the loss of the previously mentioned birds. The loss is then 12,500 hours of enjoyment, and there is still the problem of putting a value on it. In the past, economists have attempted to value recreation on the basis of travel costs, money spent on equipment, the leisure hours tradeoff against employment income, and many other methods. The potential loss of employment income probably sets the upper limit, while the casual observer, at no cost, sets the lower limit. At this stage, no attempt to put a value on leisure hours will be made.

The point to be made here is that the researchers have to provide risk data on the consequences of varying levels of toxic substances in the environment. Without the probabilities of resultant consequences being specified, no cost-benefit analysis can be performed and resource allocation cannot be optimized. Note that the incremental increases in social costs in the Legal Usage columns and the Uncontrolled Imports columns in the figures can be balanced against the same columns in the Cost of Controlling Usage subsection. Alternatively, the cost and effectiveness of sewage sludge incineration may result in a considerable reduction in the Social (Risk) Costs such that other Controlling Usage Costs may be reduced. The combinations and permutations are manifold, but operational research methods are available to find the optimum least-cost arrangement.

STRUCTURE OF TOXIC SUBSTANCE RESEARCH

In conclusion, it will be indicated how cost-benefit analysis relates to the overall scheme of things, along with a few comments about the organizational structure. The following (figure 1) shows a conical structure with the research disciplines at the base, leading up through Economic and Social Cost and Social Goals Policy to Legislation and Regulation at the apex.

It may be observed that, at present, the research

Table 4. Indirect costs of PCB's

	Risk (cost) due existing pool	Risk (cost) due continuing usage	Risk (cost) due uncontrolled imports
Cost of controlling usage			
Social costs (environmental damage costs)			
Indirect costs			
Aesthetics - loss of osprey, bald eagle, cormorant, bittern, heron, loon			
Recreational fishing			
Recreational hunting			
Cleanup of alewife dieoff			
Human - loss of physical well-being (including abortions)			
Loss of mental well-being			
Loss of social well-being			

tends to be fragmented and uncoordinated. The transportation model (second level in the cone), which shows how a particular toxic substance cycles through the natural, biological, and economic environment, tends to tie together all the various disciplines. The next level, economic and social cost assessments, should also provide a unifying force among these disciplines, as well as linking the research with the social goals.

Further, the ultimate unifying research objective may be stated as:

> To determine the tolerable level of specific toxic substances in the environment with a holistic approach that balances the direct and indirect, private and social costs and benefits relevant to the use, or nonuse of the substance.

Clearly, to meet this objective, some form of centralized body is needed in each country to integrate and coordinate toxic substance research among the various agencies and disciplines involved. These bodies would specify and insure the planning, implementation, and control strategy for achieving the research objective, including:

a. the organization and allocation of research projects and subobjectives among the various agencies;
b. the identification and effective communication of available information and data;
c. the periodic communication of the overall strategy and progress in meeting objectives; and

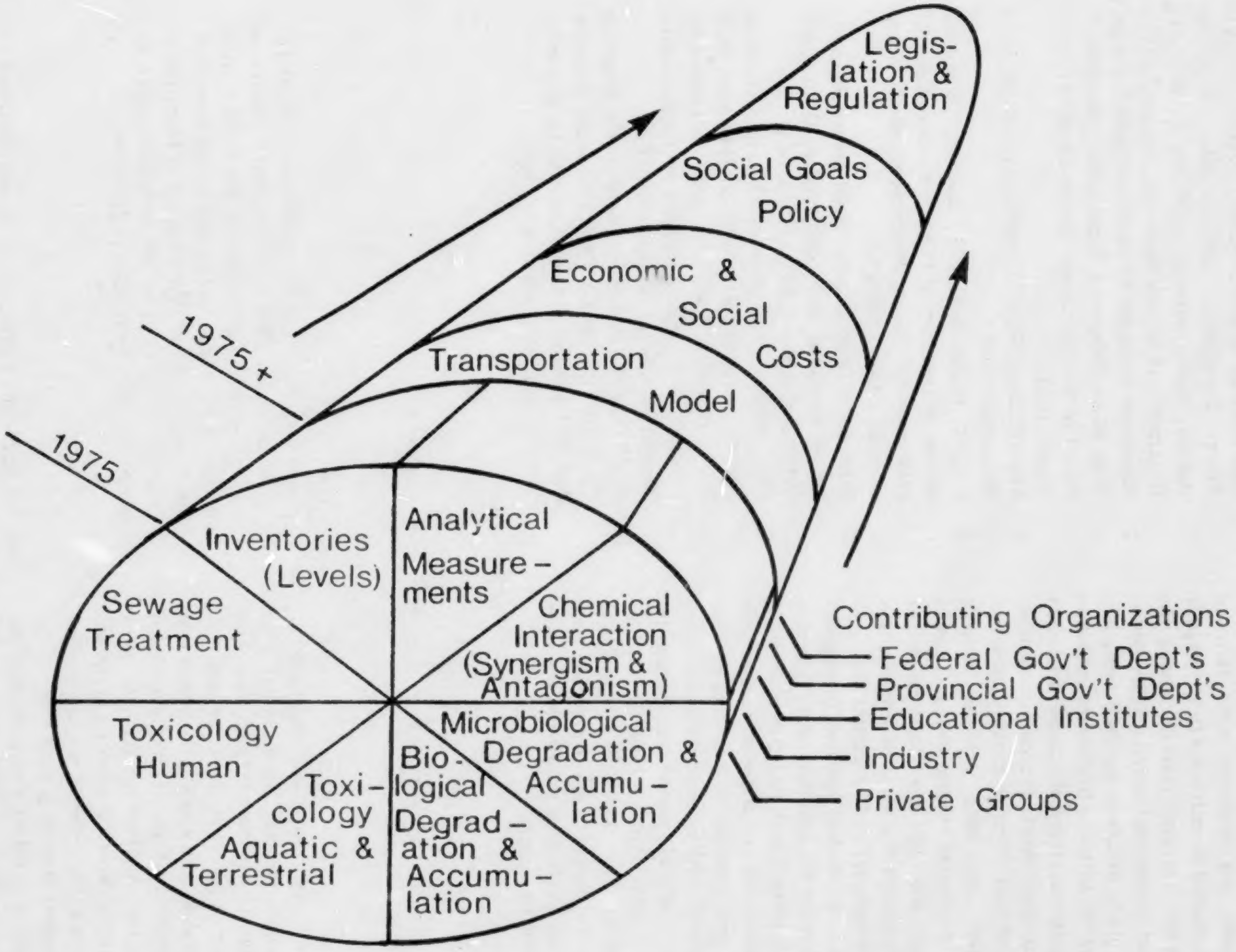

Figure 1. Toxic substances simplified function organization.

d. the formulation of recommendations based on the research findings, and their communication to policy and decisionmakers.

Clearly, to meet this objective, some form of centralized body is needed in each country to integrate and coordinate toxic substance research among the various agencies and disciplines involved. These bodies would specify and insure the planning, implementation, and control strategy for achieving the research objective, including: (a) the organization and allocation of research projects and subobjectives among the various agencies; (b) the identification and effective communication of available information and data; (c) the periodic communication of the overall strategy and progress in meeting objectives; and (d) the formulation of recommendations based on the research findings, and their communication to policy and decisionmakers.

All the elements for the lead agencies, the Department of Environment and the Environmental Protection Agency, appear to be present, such that they could grasp the authority and carry out the above objectives. It is suggested, as a first step, that each of these lead agencies appoint a Toxic Substances Research Coordination Committee. Both these national committees should be endowed with the power, through interdepartmental agreements, to carry out their objectives effectively. If we can proceed in this direction and if everyone promotes these objectives, an effective solution to the toxic substances problem will be realized.

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CHAIRMAN TIMM: I have just a few comments on what kind of comes through to me, about three things really. Hopefully it came through to you on the various types of presentations we've had. Obviously we are dealing with a very real problem that impacts the livelihood of people in this country, other countries, etc. Another big thing to come through was that the technical people and the scientific people have got to improve our communication of the scientific facts that we know to the people that are affected. I think this is probably the biggest frustration on the part of the average citizen—he just doesn't understand what we're doing or what we find out or maybe does not know what it means. But I think we've got to continue to try.

Finally, no matter what action is taken, it will have an adverse effect on some people, which of course is an impact on the environment. I think like the Senator says, EPA is in a "no win" situation as far as everybody is concerned. But I think we've learned some things tonight and gained some comments good and bad that are going to help us to go forward. Carlos wants to have some additional comments and then we'll open to the floor.

MR. CARLOS FETTEROLF: I am very disturbed by an item in the statement of the man from the Hudson Valley representing the Sloop Restoration Society. He told us that Ogden Reid, Commissioner of the New York State Department of Environmental Conservation, applauded the Canada Department of Health and Welfare for establishing its fish

PCB regulation at 2 ppm and that Reid felt the number should be 1 ppm.

I am also disturbed by the Indiana Senator urging that we all go home and pound on our legislators for action on PCB's.

There may not be one immediately correct solution to the PCB problem, but there is a best solution. The best solution is one which recognizes all phases of the problem; weighs the advantages, disadvantages, and costs; evaluates the impacts on the environment, the industry, and the people. I don't think Ogden Reid can do that off the top of his head, nor do I think a State legislature can do it without great thought and the wisest technical guidance. I'm worried. I don't want to see a panic situation. I hope USFDA does not panic. Once regulators deviate from scientifically defensible environmental actions, everyone loses. We waste resources, we trade one problem for another, and we create unnecessary costs and limitations on important segments of society. EPA must take some action. Hopefully, it will be scientifically defensible and not emotionally or politically motivated.

MR. JOHN CHASTAM (Lake Erie Cleanup Committee, Toledo, Ohio): I represent the Lake Erie Clean Up Committee, Michigan United Conservation Clubs, and the Associated Yacht Clubs of Toledo, Ohio. I wasn't going to get up to make any statement whatsoever, but after what I've heard here tonight, I feel very much like Carlos.

Several years ago, you remember taconite was a big issue after the mercury scare. Everybody was under the impression that everything was all settled and all of a sudden we had taconite. We got asbestos and that worried me.

I contacted as many of the Governors and as many of the State officials as I possibly could around the Great Lakes for the simple reason that you people out there as well as me own a part of our Great Lakes. Industry doesn't own them, individuals don't own them, they're a collective thing that we all have to enjoy. I just want you people to know that the fight is ours. Industry should realize what its doing to the very people they need for help, the people that they employ.

GENERAL CHAIRMAN BUCKLEY: I want to say about three words and they really are that I deeply appreciate your being here. Obviously I can't agree with each comment because there have been some mutually exclusive ones, but I certainly found it informative and really appreciate this. And the other thing I have is a personal observation. I think you're an enormously courteous audience and you've sat, you've listened to views that were entirely different from your own. I really enjoyed it, and thanks again.

MR. TIMM: Good night. I got roasted literally as well as physically.

COMMUNICATIONS
TO THE CONFERENCE

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SOME ADDITIONAL COMMENTS WITH RESPECT TO AMBIENT AIR SAMPLING FOR PCB'S

Gordon H. Thomas*

The ORF, under contract to the Air Resources Branch of the Ontario Ministry of the Environment, has conducted ambient air sampling for PCB's.

The ambient air was sampled for gaseous constituents by metering the air through impingers containing ethylene glycol. In addition, sampling of the ambient air for suspended particulates was performed concurrently with the impinger sampling. For this purpose, a Hi-Vol suspended particulate sampler using a glass fiber filter was employed.

Standard analytical procedures for the extraction, cleanup, and separation of PCB's from interfering components were followed. Final extracts were analyzed by gas chromatography using electron capture detection. Confirmation of the presence of PCB's in some of the extracts was obtained with the aid of gas chromatography-mass spectrometry.

PCB's were detected in both gaseous and particulate sample extracts. Levels found were as follows:

Gaseous	-	0.9 - 2.6 ng/m^3
Particulate	-	0.14 - 0.61 ng/m^3

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*Senior Research Scientist, Ontario Research Foundation, Sheridan Park, Mississauga, Ontario, Canada, L5K 1B3.

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JACK TAYLOR: Gedcor Corporation markets a solvent swellable polymer manufactured by Dow Chemical Company that possesses the unique ability to absorb or imbibe a broad range of hydrocarbons, including chlorinated ones such as askarels. The polymer, sold under the name "Dow Imbiber Bead," draws the hydrocarbon fluid into it's molecular structure, thereby capturing and containing the hazardous substance. The sealoff attribute of the bead allows valves to be manufactured that will allow water to pass and prevent free hydrocarbon escape into the environment.

MS. DORCAS THOMPSON (private citizen): I would like to speak on citizen involvement awareness and communication. It is not enough to have hearings on toxic substances, pollution problems, energy problems, transportation. Ordinary citizens feel they have much to do. They will not get interested in any problems they should be concerned about, because they are not at all aware of the problems. If a problem such as PCB's is identified, EPA must issue "educational" public releases before hearings regularly to inform people of this threat to our health. Of registered attendees at the conference there are few Chicago suburban citizens—all attendees connected with companies or agencies such as EPA. Not one suburban environmentalist or health person is registered. Several citizens such as L. W. Van Audobon are present.

If dangers are identified, we need to be educated by "decree;" signs must be posted on all areas of known PCB traces or presence—such as in air, parks, streets, buildings, beaches, water, markets, and fish and other foods. (We need to treat this similarly to the warning signs on cigarettes.) The knowledge of the presence of PCB's in breast milk must also be imparted to hospitals and new mothers—if all of these warnings were posted with addresses of persons to write, people would be concerned and would know their opinions and concerns are important. I disagree with Dr. Muir's concern over industry—he is compromising by his "philosophical" remarks. As we see Dow, Corning, and Shell have a new product (we are not so entrenched that we must consider economic factors of companies). Relocate company personnel, reeducate personnel to new job, retread personnel, computerize information, and compare notes industry to industry. After all, none of our businesses were in business 100 years ago (in the same States they are in today) so let us gear for change and not allow any industries dangerous ecologically or environmentally.

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LETTER TO CONFERENCE ON POLYCHLORINATED BIPHENYLS

Susan E. Caswell
Friends of the Earth, Madison, Wisconsin

November 19, 1975

I am sending this statement to the Environmental Protection Agency technical conference on polychlorinated biphenyls for the 200 members of the Madison, Wisconsin, branch of Friends of the Earth. We are deeply concerned about the growing PCB contamination of rivers and lakes in the Midwest and throughout the country. We are particularly disturbed by the apparent high levels of these chemicals in fish and fowl taken from Lake Michigan and from the Upper Mississippi River.

Both of these great bodies of water have been major sources for recreational and commercial fishing in the Midwest and provide the municipal water supplies for many communities. It seems to us that irreparable harm has already been done to the sport and commercial fishing industries which utilize these water resources and that a very real threat exists to the health of persons ingesting PCB contaminated fish and fowl or drinking from PCB contaminated waters.

We feel that scientific studies have proven the toxicity of PCBs to a wide variety of wildlife including primates even at levels below the standards set by the Federal Drug Administration. That they are also toxic to man has been shown by the tragic Japanese experience.

The fact that polychlorinated biphenyls are virtually indestructible by ordinary chemical or microbial processes means that they will persist in the environment for all time, and since their uses in industry are so diverse and widespread, the possibility for environmental contamination seems to us to be even more serious in the long run than the analogous situation with DDT. Indeed, there is evidence that PCBs are already ubiquitous in the environment—having been detected in the sewage of all major cities, in snow melts, in organisms from the Atlantic Ocean, as well as in fish and sediments of many lakes and rivers.

Therefore, we feel that the time to take effective action on this grave pollution problem is long past due. We favor an outright Congressional ban on production and use of PCB's, but in the meantime, we call on the Environmental Protection Agency to develop a comprehensive monitoring program for identifying sources of PCB pollution of Lake Michigan and the Upper Mississippi River as well as other lakes and rivers and immediately begin to exercise its authority under the Water Pollution Control Act of 1972 to regulate or eliminate PCB discharges. Thank you.

/s/ Susan E. Caswell
Friends of the Earth
Madison, Wisconsin

LETTER TO THE ADMINISTRATOR OF THE ENVIRONMENTAL PROTECTION AGENCY

Mrs. Meredith C. Tucker

September 8, 1975

Russell Train, Administrator
U.S. Environmental Protection Agency
Waterside Mall
Washington, D.C.

Dear Mr. Train:

We are most concerned with the lack of action the EPA is taking with regard to PCB pollution in Lake Michigan and other waterways. The seriousness of PCB pollution has been well known since 1972, yet nothing has been done to control the release of PCB's into the environment.

It seems clear that Monsanto Company is not going to voluntarily withdraw the chemical from the market; nor are imports of PCB's going to cease through informing industrial users about chemical concentrations in our water. Much stronger enforcement measures are needed.

Banning the use of PCB's clearly seems to be the only real solution to the problem. Since substitute chemicals are available for all uses of polychlorinated biphenyls, banning PCB's should present no real hardship.

As a first step toward eliminating PCB's from Lake Michigan and other water sources, we advocate participation in the National Conference on PCB's. Industrial users should be alerted to the dangers of PCB's and to alternative chemicals they could use. In addition, the public should be aware of the dangers the chemical poses in use or through human consumption of fish and water.

We urge you to take strong and immediate action to eliminate the environmental pollution of PCB's. Thank you for your attention to this matter.

Sincerely,

/s/ Meredith C. Tucker (Mrs.)
Pesticide Control Committee
Chicagoland Chapter
Friends of the Earth
498 N. Inverway Road
Inverness, Palatine
Illinois 60067

:c: Senator Percy
Senator Stevenson
Lake Mich. Fed.

LETTER TO LAKE MICHIGAN FEDERATION

Chairperson, Pesticide Committee
Knob & Valley Audobon Society of Southern Indiana

November 17, 1975

Lake Michigan Federation
53 W. Jackson
Chicago, Illinois 60604

Dear Sirs,

By unanimous decision the Knob & Valley Audobon Society of Southern Indiana voted to advocate the ban of PCB's or the severe restriction of their use. This recommendation is based on several factors:

1. The permanent nature of PCB'S. They are not biodegradable and are stable to heat making them very difficult to be destroyed.
2. The high levels of PCB's already found in fish in the Hudson River, Lake Michigan, and other bodies of water.
3. The toxic nature of PCB's to human life.

If possible please have our position presented at the EPA conference in Chicago.

Sincerely,

/s/ Chairperson, Pesticide Committee
Box 237
Lanesville, Indiana 47136

LETTER TO THE ENVIRONMENTAL PROTECTION AGENCY, REGION V

Douglas V. Whitesides, Jr.

November 16, 1975

U.S. – EPA, Region V
Chicago, Illinois

Re: PCB Problem Conference
November 19-20-21, 1975, Chicago, Illinois

Gentlemen:

Due to the buildup of PCB's (polychlorinated biphenyls) in the environment and the serious problems caused by this material, I believe that its manufacture and use should be completely banned immediately.

Surely the world will be a much better place without this extremely environmentally degrading substance.

Thank you,

/s/ Douglas V. Whitesides
Rt. 1, Box 296
Lanesville, Indiana 47136

INTRODUCTORY REMARKS

John L. Buckley, Ph.D.

I would like to call to order the final morning of our conference here and I would like to tell you also that I have won an additional brass ring. I will be chairman of the session this morning, not because I planned it that way, but because Dr. Jim Brydon, who had agreed to serve in this capacity and did his best to get here, could not make it. He left Ottawa late yesterday afternoon, having appeared before the Treasury Board in the morning and the Senate in the afternoon, and he made it as far as Toronto, where apparently there were electrical difficulties and so Jim will about this time be turning around to go back to Ottawa instead of coming here.

I would like to explain to you what I have in mind in the way we are going to try and operate. It is not all that difficult or complex. It rather follows the statement in the program. We will have a series of speakers, each of whom has prepared remarks and that will be followed by discussion among the speakers and in response to comments or questions from the floor.

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A REVIEW OF FEDERAL AND STATE GOVERNMENT ROLES IN CONTROLLING IMPACTS OF PCB's ON THE ENVIRONMENT

A. Karim Ahmed, Ph.D.*

Abstract

Polychlorinated biphenyls (PCB's) have found extensive application in a large number of industrial products since they were first commercially used in the 1930's. Presently their use in the United States appears to be principally restricted to closed electrical systems, such as capacitors and transformers, though several tens of millions of pounds are used each year as an insulating fluid in these products. Numerous studies now clearly indicate the contamination of PCB's in the environment. Residues of PCB's are detected in many different aquatic and animal species. They are found in high concentrations in river bottoms, and in samples of commercial and sport fish collected from the Great Lakes region and in several rivers in the East Coast. They are found in amounts greater than the Food and Drug Administration's tolerance limit of 5 ppm.

The roles of the Federal and State governments in controlling the manufacture and use of PCB's are examined. It is concluded that, in most cases, little or no regulatory activities have been initiated by government agencies in recent years to address the problem, in spite of the heightened concern over the potential threat of PCB's on the environment and to public health. Only in the past few months have several Midwestern States bordering the Great Lakes and the State of New York taken measures to control the discharge of PCB's from known sources.

To cope with the problem, several recommendations have been urged, which include: the complete phaseout of the manufacture and use of PCB's, a ban on their import and export, the development of a consumption inventory, an accelerated program of monitoring and surveillance, a moratorium on river bottom dredging, promulgation of the PCB toxic effluent standard, a significant lowering of present FDA tolerance limit on PCB's, and Congressional passage of the Toxic Substances Control Act.

INTRODUCTION

Polychlorinated biphenyls (PCB's) were first introduced into commercial use over 45 years ago, and for a long time they were considered to be relatively nontoxic substances. Their potential threat to the environment was not recognized until 1966, when the Swedish scientist, S. Jensen, observed the presence of PCB's in fish and wildlife samples while analyzing for chlorinated hydrocarbon pesticide residues (refs. 1,2). Since that initial discovery, PCB's have been detected in numerous aquatic and animal species, and have been noted in high concentrations in industrial waste discharges, river bottom sediments, food packaging materials, and in food products, such as poultry, fish, and dairy products.

A large variety of industrial applications have been found for PCB's, since they are endowed with a number of desirable chemical and physical properties. They are chemically stable, nonflammable, and essentially nonsoluble in water. They also possess a high dielectric constant and are relatively viscous materials with a low volatility. Thus, they are used as heat-exchange fluids and as a dielectric medium in electrical capacitors and transformers. They have been used as hydraulic and lubricating fluids, and are used in gas turbines and vacuum pumps. In the past, they were extensively used as plasticizers in plastic products, as coatings in textile products, and in paints and varnishes. They were also used as sealants, as extenders in pesticides, and as an ingredient in caulking compounds, adhesives, printing inks, and carbonless duplicating paper (refs. 3,4,5).

In 1971, over 106 million pounds of PCB's were produced in the industrial countries of the world, with a third or nearly 40 million pounds being manufactured in the United States (ref. 4). During the late 1950's and into the 1960's, U.S. domestic sales of PCB's increased nearly threefold, reaching a peak of approximately 72 million pounds in 1970 (ref. 5). Prior to 1970, about 60 percent of U.S. domestic sales was for closed electrical systems, such as capacitors and transformers. Another 25 percent was used as plasticizers and in the production of carbonless duplicating paper (ref. 5). Since September, 1970, when Monsanto Company, the sole manufacturer of PCB's in the United States, restricted its sale of PCB's to closed electrical systems, the U.S. consumption has been reduced considerably. It is important to note, however, that these reductions occurred mainly with the use of PCB's as plasticizers, and as hydraulic and lubricating fluids and other miscellaneous uses. The bulk of PCB's, as mentioned above, had been used in electrical systems, and it is still being used almost exclusively by manufacturers of large power and small ballast capacitors, and electrical transformers, as the preferred dielectric fluid.

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*Staff Scientist, Natural Resources Defense Council, New York, New York.

ENVIRONMENTAL CONTAMINATION OF PCB's

Residues of PCB's in environmental samples have now been reported in numerous studies conducted during the past 10 years. They have been analyzed in wildlife and fish samples in Holland, Germany, and in the Baltic Sea (refs. 6,7). Off the coast of Scotland, PCB residues were found in several marine species, the contamination presumably having arisen from sewage wastes being dumped into the open sea (ref. 8).

Unlike the almost ubiquitous presence of chlorinated hydrocarbon pesticides in the environment, PCB residues are generally found in areas associated with high industrial and urban activity. Little or no detectable amounts of PCB's were found in wildlife sampled in remote, nonpopulated areas, such as the Gulf of California and Cape Crozier, Antarctica (ref. 9). Similarly, rivers and lakes without known industrial discharges are generally found to be uncontaminated with PCB's, such as has been shown for several watersheds and small lakes in the upper Midwest and in Canada (refs. 10,11). On the other hand, PCB's in increased amounts are detected in the Eastern and Western coastal regions in highly urbanized areas, such as the Chesapeake Bay, San Fransisco Bay, and the Puget Sound, or in regions where industrial discharges are known to occur, such as Escambia Bay, Florida, and the Hudson River (refs. 9,12,13,14).

Studies on plankton species in the marine environment have shown them to contain detectable amounts of PCB's. Zooplankton sampled off the Eastern continental shelf area were observed to contain PCB levels ranging between 0.07 and 3 ppm (ref. 15). Plankton species collected in the Gulf of Mexico and the Gulf of St. Lawrence have been found to contain PCB residues as high as 1 ppm and 3 ppm, respectively (refs. 16,17).

Tuna caught off the Atlantic seaboard are shown to contain PCB's with levels of 0.36 to 1.5 ppm (ref. 18). Considerably high concentrations of PCB's are noted in several predatory birds, with values from 30 to 900 ppm in the livers of heron, 65 ppm (wet weight) in peregrine falcons, 190 ppm (whole body weight) in white-tailed eagles, to 93-470 ppm (wet weight) in the livers of cormorants (ref. 8, 9, 19, 20). Similarly, a number of studies have shown relatively high levels of PCB's in the eggs of wild birds, ranging from 1 to 44 ppm (refs. 18, 19, 21).

An important impact of PCB's on the environment has been contamination of sport and commercial fisheries in the Great Lakes region. Levels of PCB's in lake trout and coho salmon from Lake Michigan show an alarmingly increasing trend since 1972, in the average range of 10 to 25 ppm, well above the present Food and Drug Administration's maximum allowable concentration of 5 ppm for fish and shellfish (see figure 1) (ref. 22). On the other hand, DDT levels for the same species of fish show a decreasing trend since 1970, when DDT consumption in the United States was curtailed (figure 1). It is rather discouraging to note that even with the restriction of domestic sales of PCB's to closed electrical systems since the early 1970's there has been no corresponding decrease in PCB levels in commercially important fish species.

Fish samples collected by the Wisconsin Department of Natural Resources last year showed extensive contamination of several species of fish in Lake Michigan (ref. 23). These include chinook salmon, coho salmon, brown trout, tiger trout, lake trout, whitefish and carp. Generally, the residue levels ranged well above 5 ppm, with lake trout (up to 43.8 ppm) and carp (up to 51.6 ppm) showing the highest concentration values. In the Upper Fox River fish sampled at one station earlier this year by the Department showed high concentration of PCB's in white suckers (32.7 ppm), carp (21.4 to 45.8 ppm, average 35.9 ppm) and northern pike (average 15.4 ppm). Samples analyzed by the Department in 1973 showed similar residues of PCB's in a number of fish species in several reaches of the Mississippi River. These data are summarized in table 1.

An interagency Task-Force on PCB's, formed of Federal and several State agencies of Minnesota and Wisconsin, sampled fish in the Mississippi River during the early summer of this year and found similar levels of PCB's in several fish species: carp (up to 33 ppm), walleye (up to 9.8 ppm), white bass (up to 4.3 ppm). Generally, PCB values were found at higher levels in samples collected south of the Minneapolis-St. Paul metropolitan area (ref. 24). More surprising is the detection of PCB residues in lake trout sampled last year in Lake Superior by the Great Lakes Environmental Contaminants Survey (GLECS), which showed residue levels ranging between 0.5 and 12.7 ppm in the Siscowet (fat) variety of trout. The lean trout variety had lower PCB residues, though the larger size fish often had residue levels close to or above 5 ppm (ref. 25).

The Division of Fish and Game of the Commonwealth of Massachusetts has in the past occassionally sampled PCB levels in fish in several rivers and streams in the State (ref. 26) and high levels have been detected in several species. White sucker samples collected in 1971 in the Housatonic River showed mean PCB levels of 15.10 and 69.30 ppm at two sampling stations near the General Electric plant in Pittsfield, Massachusetts, which

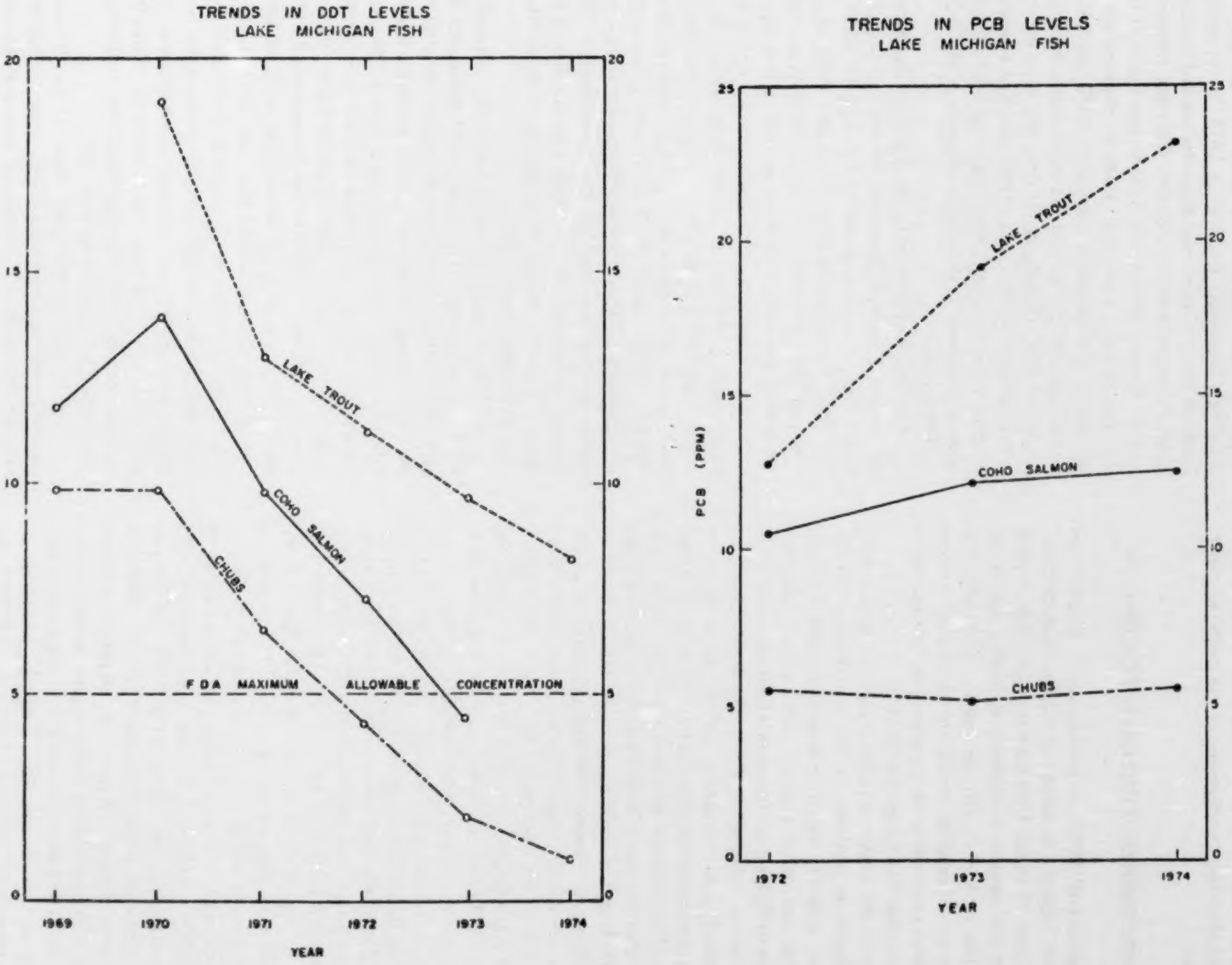

Figure 1. PCB and DDT trends in Lake Michigan fish (ref. 22).

Table 1. PCB's residue in fish species in Mississippi River, 1973 (ref. 23)

Species	Station	PCB(ppm)	PCB average
Walleye	Lake Pepin	11.9 - 31.4	21.9
	Prescott	7.21 - 11.1	9.04
	Wabasha	3.84 - 9.00	5.85
	Trempealeau	3.16 - 6.34	4.98
Largemouth bass	Lake Pepin	1.65 - 10.5	4.90
	Trempealeau	1.74 - 5.92	2.94
	La Crosse	1.49 - 4.24	2.62
Northern redhorse	Lake Pepin	3.45 - 8.53	5.94
	Prescott	1.01 - 16.2	5.71
Carp	Lake Pepin	5.49 - 11.2	7.69
	Prescott	0.57 - 20.44	8.36
	Wabasha	4.31 - 10.5	7.83
	La Crosse	1.95 - 4.05	2.94

manufactures electrical transformers. Though it was determined by the State agency that the contamination of the fish was due to discharge of PCB's from the General Electric plant, there has been no further monitoring of the Housatonic River since 1971, and there is no indication whether abatement procedures used by the plant were effective. From the data collected by the agency, it is also clear that in 1972 there was extensive contamination of a large variety of fish species in nearly all the rivers that were sampled in the State, with mean PCB residue levels ranging between 1.11 and 197.0 ppm (see table 2).

To date, the most contaminated fish sampled in this country are found in the upper reaches of the Hudson River, near and below two General Electric capacitor plants in the Fort Edward-Hudson Falls area. Earlier sampling by the Federal EPA last year had shown yellow perch and shiner minnows to contain average PCB residues of 17 and 78 ppm, respectively, with one rock bass sample containing 350 ppm (ref. 27). It appears that the two General Electric plants had been discharging approximately 30 pounds of PCB's per day into the river, though in recent months there appears to have been a reduction of daily discharges to less than 10 pounds. The most recent fish samples collected by the New York State Department of Environmental Conservation confirm the earlier findings. The composite PCB averages of fish caught near the plant show typical values well above 20 ppm, with one American eel sample yielding residue values of 403.4 ppm of Aroclor 1016/1242 (ref. 28). These findings are partially summarized in table 3.

At the same time, the New York State Department of Environmental Conservation sampling of fish species of recreational and commercial importance in the lower Hudson also shows high residues of PCB (ref. 28). Striped bass caught near the Albany area showed total PCB residue values ranging between 11.08 and 89.76 ppm. PCB residues in fish caught in the lower reaches of the river have higher concentrations of Aroclor 1254, indicating long-range transport of the previously used higher chlorinated mixture of PCB discharged by industrial activities in the past. Striped bass sampled near the West Point and Tappan Zee area of the Hudson River (50 miles north of New York City and several hundred miles south of the Fort Edward-Hudson Falls area) show total PCB residues ranging between 1.16 and 7.54 ppm. Similarly, a sample of American shad in the Poughkeepsie area showed PCB residue levels of 9.0 ppm.

Of equal concern is the accumulation of PCB in sediments of the river bottoms. PCB's, like most chlorinated hydrocarbons, are highly insoluble in water and tend to settle on river sediment quite readily, being adsorbed on silt and fine particles in river bottoms (ref. 12).

Data collected by the United States Geological Survey (USGS) on the Hudson River during the years 1973-74 show the presence of PCB's in the water and in the bottom sediments of the river (ref. 29). Values ranging between 0.3 and 3.0 ppb PCB have been reported in water samples collected at several stations on the Hudson River. These include locations near Poughkeepsie, Waterford, Chelsea, and Rhinebeck.

Table 2. Mean PCB concentration (ppm, dry weight) in fish collected in Massachusetts rivers and streams (ref. 26)

Stream and station	Town	No. and species	Aroclor (ppm)	Rank 1972	Rank 1971
		Aroclor 1248			
Millers	Athol	1 White sucker	197.0	1	[a]
Deerfield	Deerfield	5 Fallfish	23.8	2	5
Merrimack #1	Tyngsboro	5 Pumpkinseed	19.1	3	-
Connecticut	Northampton	5 Fallfish	17.7	4	6
Merrimack #2	Haverhill	3 Alewives 1 Eel 1 Banded killifish	13.3	5	4
Chicopee	Ludlow	5 Pumpkinseed	13.3	6	2
Westfield	Westfield	5 Pumpkinseed	11.9	7	3
Blackstone	Millville	5 White sucker	10.1	8	1
Little	Westfield	5 Golden shiner	9.41	9	7
Ware	Thorndike	5 Pumpkinseed	3.32	10	-
		Aroclor 1260			
Blackstone	Millville	5 White sucker	21.3		
		Aroclor 1254			
Charles #2	Cambridge	5 Pumpkinseed	8.3	1	1
Concord	Concord	5 Bluegill	5.96	2	2
Taunton	Taunton	5 Bluegill	3.13	3	3
Quaboag	Palmer	5 Redbreast sunfish	1.11	4	-

[a] No fish could be collected in previous years.

On the other hand, PCB concentrations analyzed from the bottom sediments of the river showed considerably higher values characteristic of the insoluble nature of PCB's. The USGS data are summarized in table 4.

More recently, the U.S. EPA conducted water and sediment analysis for PCB's in the vicinity of Fort Edward, near the GE plant. Water samples collected immediately south of the GE outfalls had PCB values of 2.2 to 3.1 ppb. The sediments contained 540,000 to 2,980,000 ppb (540 to 2,980 ppm) PCB, with 6,600 ppb being detected several miles downstream of the discharge pipes (ref. 27).

These very high concentrations of PCB's in the sediments of the Hudson River for which dredging is proposed are particularly troubling in light of the fact that the highest concentrations are likely to exist in the deep navigation channel. PCB is associated more with finer grain particles than with particles of larger mean grain-size diameters. This was clearly observed in extensive studies carried out in a study conducted by the State of Maryland Department of Natural Resources in conjunction with Westinghouse Electric Company in the Chester River (ref. 12). The study concluded as follows:

> Laboratory and field studies by other workers as well as the present study have indicated that the relatively water-insoluble chlorinated hydrocarbons readily adsorb to the surfaces of suspended clay minerals, and, in turbid waters, are found mainly attached to the suspended

Table 3. Composite PCB residue in fish species sampled near the Fort Edward-Hudson Falls area of the Hudson River (ref. 28)

Species	Station	PCB, ppm (Aroclor 1242/1016)
Rock bass	Fort Edward	27.35
Walleye	Fort Edward	104.21, 81.12, 9.88
	5 miles below GE	157.27
Yellow perch	Fort Edward	61.10,236.4, 128.3
	5 miles below GE	84.13, 35.5, 88
White sucker	Fort Edward	82.9, 8.8
	5 miles below GE	51.39, 29.7, 36.03, 78.0, 57.5
	Stillwater	10.3, 154.3
	Waterford	20.3, 36.0
Brown bullhead	5 miles below GE	73.24, 85.30
Largemouth bass	Stillwater	24.85, 14.31, 8.99
Smallmouth bass	Waterford	27.0, 36.0

Table 4. PCB's concentration in Hudson River sediments, USGS (ref. 29)

Date	Station	PCB in sediments (ppb)
7/74	Chelsea	3,200
9/74	Chelsea	1,800
7/74	Poughkeepsie	11,000
9/74	Poughkeepsie	3,600
7/74	Waterford	13,000
9/74	Waterford	640
8/75	Roger's Island, south of Glen Falls	18,000
7/74	Winebrook Hills	0.1

particulate matter. In the Chester River Study, *the inverse correlation between mean grain-size diameter of the sediments and chlorinated hydrocarbons content* [including PCB] *suggest that a major route of transport of these compounds into the Chester River is attached to the suspended sediments* that sweep into the lower river from Chesapeake Bay. The sediments are distributed in the bed of the Chester River in accordance to their grain size and the local current velocity regimes. The fine-grain materials (silts and clays) are thrown into suspension in the shallow, shoreline areas by wind-driven and tidal currents. *These materials tend to collect in the deeper channels of the river, and, as is suggested by the core data, the fine-grain sediment areas in the deeper channels and submerged terraces probably represent the major "sinks" for chlorinated hydrocarbons* in the Chester River. (Emphasis added.)

The above observation of the transport and adsorption properties of several chlorinated hydrocarbons, including PCB's, has been further confirmed by a recently completed study in the Upper Chesapeake Bay area (ref. 30). Thus, it has been abundantly demonstrated that not only are PCB's adsorbed on fine-grain particulates, but they are also found in highest concentrations in the deeper channels of the river, where the channels act as "sinks" for the insoluble hydrocarbons. Thus, the very areas where dredging operations are being proposed are the most contaminated.

TOXICITY OF PCB's–A BRIEF APPRAISAL

Effects on Aquatic Species

PCB's, as a class of compounds, are extremely persistent and nonbiodegradable substances, and tend to bioaccumulate in the aquatic environment by factors of a few thousand-to several hundred-thousand-fold. PCB's (Aroclor 1254) have been shown to concentrate over 20,000 times the water levels in crustacea, 10,000-to 50,000-fold in certain estuarine fish species, to as much as 200,000-fold with fathead minnows (Aroclor 1242 and 1254) (ref. 31).

The most serious effect of PCB's on aquatic species is their ability to interfere in reproductive process and hatchability of fish eggs. It appears that the thresholds for whole salmon egg mortality is about 0.5 ppb (ref. 32). Similarly, the production and hatchability of fathead minnow eggs was adversely affected by 0.9 ppb of Aroclor 1254 (ref. 33). Pinfish and spot were unable to survive at 5 ppb of Aroclor 1254 (ref. 34) and many aquatic invertebrates were clearly affected at concentration ranges of 1 to 10 ppb PCB, including blue crabs, pink shrimp, and oyster (ref. 14). Studies by Hansen and his colleagues at EPA's Gulf Breeze Environmental Research Laboratory have indicated comparable mortality to several aquatic species exposed to Aroclor 1016 (ref. 35). These findings show that acute 96-hour LC_{50}'s (or EC_{50}'s) for oyster, brown shrimp, and grass shrimp were 10.2 ppb, 10.5 ppb and 12.5 ppb, respectively. It thus appears that the substitution of the lower chlorinated homolog mixture, Aroclor 1016 (chlorine content, 41%), does not diminish the impact of PCB's on the aquatic environment. Based on the high lethality of PCB's to marine and freshwater aquatic species, the National Academy of Sciences-National Academy of Engineering Committee made the following recommendation:

> Aquatic life should be protected where the maximum concentration of total PCB in unfiltered water does not exceed 0.002 microgram per liter [0.002 ppb] at any time or place, and the residues in the general body tissues of any aquatic organism do not exceed 0.5 microgram per gram [0.5 ppm] (ref. 31).

Effects on Humans and Animals

The toxicity of PCB to humans has been clearly demonstrated. In 1968 a severe form of skin disease was diagnosed in Japanese families that consumed rice oil contaminated with Kanachlor 400 (PCB with 48 percent chlorine content) (refs. 36,37). A total of 1,057 poisoning cases have been recorded to date in Japan (ref. 38). The disease is characterized by swelling of the upper eyelids, visual impariment, acne-like formations, and heightened pigmentation of the skin (ref. 36). Patients with this disease also exhibited neurological disorders and showed signs of hearing loss. Several babies born of women patients had many of the symptoms of the disease, which indicated a placental transport of PCB, and most fetuses were born smaller than the national average. Another disturbing feature of this disease was the observation that most patients recovered very slowly, which suggested that PCB was retained in the human body over a very long period (ref. 38).

It should be noted here that human contamination with PCB's is not isolated. PCB's have been detected in human adipose tissue in such widespread occurrence that over 40 percent of the U.S. population contains 1 part per million (ppm) or more (ref. 39).

A number of studies have conclusively shown the effect of PCB's on the reproduction of several animal species. At 100 ppm in the diet of experimental rats, PCB (Aroclor 1242 and 1254) caused a reduction in the number of offspring that were born (ref. 36). Similarly, in another study, dietary levels of as little as 20 ppm of Aroclor 1254 caused a decreased number of litters to be born to female rats (ref. 36). In a reproduction study carried out with minks, dietary levels of less than 1 ppm of PCB's caused a marked depression on the ability of minks to reproduce (ref. 40).

The effects of PCB on the reproduction of pheasants and chickens have also been demonstrated. Aroclor 1254 has a noticeable effect on both the production and hatchability of pheasant eggs (ref. 41). With chickens, dietary levels of as little as 10 ppm of Aroclor 1242 and 100 ppm of Aroclor 1254 caused thinning of eggshells, reduced egg production, and reduced hatchability (ref. 42). In this context, it is important to note that homologs of PCB with lower chlorine content may have a greater effect on reproduction than biphenyls with higher chlorine content. In a recent review article on the toxicology of PCB, Dr. Renate Kimbrough of the Center for Disease Control in Atlanta, Georgia, stated:

> The results obtained from the reproduction studies in birds particularly seem to indicate that

the lower chlorinated biphenyls affect reproduction more than the higher chlorinated biphenyls, and it is very important to determine whether the lower chlorinated biphenyls that have been suggested as replacements for presently employed chlorinated biphenyls have an effect on reproduction (ref. 36).

In an extensive series of studies carried out by Dr. James Allen and his colleagues at the University of Wisconsin Medical School, PCB's fed in the diet of rhesus monkeys caused significant effects that mimicked both the symptoms of known human toxicity and reproduction failures in experimental animals. When female rhesus monkeys were given 25 ppm of Aroclor 1248 for a period of 2 months, severe symptoms of facial swelling and skin eruptions were manifested (ref. 43). At lower dietary levels of 5 ppm and 2.5 ppm (Aroclor 1248), similar clinical symptoms were observed after a few months, and more significantly, these low levels of PCB caused marked effect on reproduction. After three matings with male monkeys, only 12.5 percent of the 5 ppm PCB dietary group and 37.5 percent of the 2.5 ppm group were pregnant as compared to 90 percent pregnancies with a comparable control group (ref. 44). At the same time, fetuses that were born were significantly smaller in size and continued to ingest high PCB levels from the nursed milk of their mothers (ref. 45). These effects at low PCB levels are particularly important to note, since the present Food and Drug Administration tolerance limit for PCB residue in fish and shellfish is 5 ppm (ref. 46), and were based on an assumption of a higher no-effect level.

Of serious concern is a recent study conducted by a group of research investigators at the Center for Disease Control, Atlanta, Georgia, the U.S. Environmental Protection Agency, the National Cancer Institute, and the Johns Hopkins University School of Medicine (ref. 47). They observed that when Sherman strain female rats were fed 100 ppm of PCB (Aroclor 1260) for about 21 months, 26 of 184 of the experimental animals examined had malignant liver tumors (hepatocellular carcinoma), whereas only 1 of 173 control animals had the same disease. At the same time, 146 of the experimental and none of the control animals had tumorous lesions (neoplastic nodules) in the liver.

Other toxic effects of PCB that have been reported in the scientific literature include: (1) increase in the biological activity of certain hydroxylating enzymes in the liver and increased activity in the rat; (2) induction of liver porphyria in several animal species; (3) interference with the immune defense mechanisms of rabbits; (4) neurological disorders in rats; (5) increased PCB residue in fatty tissues, serum, and milk; and (6) teratogenic effect in the chick embryo (refs. 48, 49).

IMPACT ON RECREATIONAL AND COMMERCIAL FISHERIES

One of the most illustrative examples of the impacts of PCB contamination on the environment is an examination of its effect on sport and commercial fisheries on the Hudson River. The Hudson River is a productive breeding ground for fish, not only for resident species such as white perch, but also for migratory oceanic species like striped bass, shad, and herring. The shortnose sturgeon, which has been classified as an endangered species, is also an inhabitant of the Hudson River (ref. 50).

Striped bass are the most economically important of the fish that spawn in the Hudson since the species supports a major recreational and commercial fishery. For that reason, too, it is a species of particular concern because of the high levels of public consumption of Hudson River striped bass and the economic dependence of commercial fishermen on the species.

The weight in 1970 of the striped bass commercial catch in the New England Region plus New York State (including the Hudson River) was 2,780,000 pounds (ref. 51). The weight in 1970 of the striped bass sport catch in the North Atlantic (which includes New York State) was 45,844,000 pounds (ref. 52). Hudson-spawned striped bass are a major component of the Atlantic stock. Though estimates have varied, there is no doubt that the contribution is substantial. The staff of the Nuclear Regulatory Commission (ref. 53) has taken the position that:

1. The Hudson River stock is the major source of striped bass caught in the Hudson River, the western half of Long Island Sound, and the New York Bight (Barnegat Inlet, N.J., to Moriches Inlet, N.Y.). In this "Inner Zone" of influence the NRC staff estimates that more than 50 percent of the sports and commercial landings are made up of Hudson-spawned striped bass and uses a figure of 90 percent for its own estimates (ref. 53).

2. In the "Outer Zone" of influence, extending from Maine to Cape May County, N.J. (less the Inner Zone), Hudson-spawned striped bass constitute somewhere between 10 and 50 percent of the sports and commercial fishery.

The average annual commercial catch of striped bass for the years 1961-1969 in the Inner Zone was 268,200 pounds (ref. 53). Using the NRC's 90 percent figure, 241,400 pounds represent the average annual commercial catch in the Inner Zone attributable to Hudson River striped bass. The average annual commercial catch in the Outer Zone is 1,969,000 (ref. 53), 50 percent of which is 984,500 pounds. The annual total for commercial catch of Hudson River striped bass is therefore estimated to be about 1,200,000 pounds.

In 1970, a pound of striped bass would bring a commercial fisherman $.26/lb on the average. The retail price would be, at a minimum, three times that amount. Striped bass is now selling at $1/lb in New York. Using this retail value of $1/lb, the value of commercial catch of Hudson River striped bass appears to be in the range of $1.2 million per year.

In 1970, the striped bass sports catch exceeded the commercial catch in the North Atlantic (which includes New York State) by a factor of 16.5 (ref. 53). Assuming an annual commercial catch of 1,200,000 pounds of Hudson River striped bass, one can extrapolate to a sports catch of 21,200,000 pounds. This amount represents a little less than 50 percent of the 45,844,000 1970 sports catch of striped bass in the North Atlantic and, therefore, is, if anything, an underestimation of the Hudson River sports catch. In sum, something on the order of 22,000,000 pounds of Hudson River striped bass are probably consumed each year, if we combine these commercial and sports catch figures. Moreover, the problem is even more serious than the above information would indicate. The striped bass is a migratory fish with stock from various sources (e.g., the Chesapeake, Delaware, and Hudson) intermingling. Since there is no way to distinguish origin of a striped bass, the entire North Atlantic stock may be contaminated.

An indication of the magnitude of the total Great Lakes fishery problem from PCB contamination can best be seen by evaluating the value of sport and commercial fisheries for the State of Michigan. The sport fisheries in the Great Lakes are comprised of coho and chinook salmon, steelhead, brown and lake trout. This fishery is known collectively as the salmon fishery. This is the fishery most substantially effected by PCB contamination.

Value of sport fishery in Michigan is determined by multiplying the number of angler days by $15 per day. In the Great Lakes contiguous with the State of Michigan in 1974, there were 3.1 million angler days devoted to the anadromous and Great Lakes fishery. The total value of this sports fishery is estimated at $46.5 million. Another determination is a total value of the fishery to resident and nonresident fishermen. Michigan has determined this to be $24 to $30 million—a somewhat lesser figure but still quite substantial. The value of the total Great Lakes sport fishery would be substantially larger when data from other States are included (ref. 54).

The commercial fishery in the Great Lakes has been on recent decline due to overfishing, so the value is substantially less than the sport fishery. In 1974, the number of pounds caught totalled 14,524,079, with an estimated value of $3,814,840.

The commercial species most affected by PCB contamination has been observed to be chubs. These fish are caught commercially in Lake Michigan and Lake Superior. Their 1974 catch and value are recorded in table 5.

As the above table indicates, chubs represent approximately 32 percent and 54 percent of the commercial fishery, respectively, in these two lakes. Any significant decrease in the availability of chub population to the commercial fishery will have a negative impact on the fish-related economy of the region. It is possible that a decreased chub population would eliminate commercial fishery from the Great Lakes altogether.

FEDERAL AND STATE GOVERNMENT ROLES

Since the early 70's, when it was increasingly realized that widespread contamination of PCB's had occurred in this country, a number of events highlight the role that the Federal and State governments have taken, or have failed to take, in addressing this problem. A Federal Interdepartmental Task Force was convened in 1972, which concluded that because of the highly persistent nature of PCB's and their bioaccumulation in the food chain, they posed a serious threat to human health. They recommend restricting the use of PCB's to closed electrical systems, such as electrical transformers and capacitors (ref. 56). However, as mentioned before, Monsanto Company had begun restricting its domestic sales for such exclusive purposes since the fall of 1970, and it is not clear what the Task Force's recommendations issued in May, 1972, had to do with the voluntary move made earlier by the company as has been suggested in recent remarks by an official of a Federal agency (ref. 57).

In July, 1973, the Federal Food and Drug Administration established tolerance limitation in a variety of food products as follows (table 6):

Toxicity data obtained during the past year that show severe reproductive failure in low concentrations of PCB in primates (5 and 2.5 ppm), and the recent demonstration of carcinogenicity with Sherman strain rats (refs. 44,47), clearly indicate the need to substantially revise the present FDA tolerance limitations of PCB's in foods. This in turn will have the effect of completely changing the definition of "unacceptable" levels of PCB's in fish and poultry, to be at times almost all

Table 5. 1974 commercial fish catch and value in Lakes Michigan and Superior (ref. 55)

	Chubs		Total Fish	
	Catch (lbs)	Value ($)	Catch (lbs)	Value ($)
Lake Michigan	1,154,067	780,793	10,924,961	2,496,208
Lake Superior	1,011,234	464,499	1,529,736	826,592

Table 6. FDA tolerances for PCB in food and food packaging (ppm) (ref. 46)

Product	Tolerance level (ppm)
Milk[a]	2.5
Dairy product[a]	2.5
Poultry[a]	5.0
Fish and shellfish[b]	5.0
Eggs	0.5
Infant and junior food	0.2
Complete and finished animal feed	0.2
Animal feed components	2.0
Paper food-packaging material	10.0

[a] On fat basis.

[b] Edible portion.

inclusive of many species that inhabit the polluted lakes, streams, or feedlots, in this country.

In spite of the deficiencies of the present tolerance standards, the FDA has in recent months taken seizure action against several fish shipments earmarked for commercial markets in Lake Michigan, since they exceeded the present 5 ppm guideline (ref. 61). Similarly, the FDA seized a shipment of carp this summer in Lake Pepin on the Mississippi River (ref. 58). In view of a recent legal decision that clearly allows the FDA to define pesticide residues in foods as an indirect food additive, the FDA is expected to enforce its guidelines more forcefully in the future. FDA officials in the Great Lakes region have warned that chubs, coho salmon, and lake trout may be disallowed in interstate commerce, since they continue to contain high residue levels of PCB's (ref. 61). It is apparent that with a more thorough monitoring and surveillance program, nearly all species of fish in the Great Lakes region and in several rivers in the East coast area will be essentially unavailable for human consumption for many years to come.

One of the great ironies of the present situation is that *over 3 years ago* the Federal Water Pollution Control Act (FWPCA) directed the Federal Government, specifically the Environmental Protection Agency (EPA) to take swift action to reduce the discharge of PCB's and other toxic water pollutants to safe levels. The fact that today there is no meaningful national program to regulate PCB discharges is a result of the Federal Government's disappointing failure to carry out the FWPCA as Congress wrote it or, for that matter, to take any other strong regulatory action.

The Congressional authors of the FWPCA believed that the "hazards posed by toxic substances" made their effective regulation "especially urgent" (ref. 59). Accordingly, they established in the act a "national policy that the discharge of toxic pollutants in toxic amounts be prohibited" (ref. 60) and added to the act a far-reaching special provision, Section 307(a), to insure that this goal was fully implemented. If Section 307 (a)'s mandatory deadlines had been met, toxic pollutant effluent standards covering PCB's and other toxic pollutants would have been promulgated by EPA roughly 2 years ago and complied with by dischargers about 1 year ago. Yet, to date EPA has failed to promulgate a single standard under this provision.

State officials have also expressed dismay over the EPA's failure to promulgate toxic substances effluent standards. John Hesse of the Michigan Department of Natural Resources in recent comments at a regional convention, stated:

> I want to mention that the delay in this standard [effluent standard for PCB] has weakened the states attack on industrial sources and is long over-due. (ref. 61)

It is instructive to review the events leading to the current situation. Section 307(a) of the act requires EPA to first develop a list of toxic water pollutants. For the substances on this list EPA must subsequently develop and enforce strict effluent standards, standards which must protect the users of the waters with an "ample margin of safety" (ref. 60). EPA began inauspiciously by failing to meet FWPCA's January 12, 1973, deadline for publication of the list of toxic pollutants. When 4 months later the list had still not been published, the Natural Resources Defense Council brought suit challenging EPA's default. That suit ended on June 19, 1973, when the U.S. District Court ordered EPA to publish the list "on or before August 31, 1973" (ref. 62). The list published by EPA as a result of this court directive, though inadequate in many respects, did include PCB's along with eight other pollutants.

Section 307(a) also requires that prior to promulgating final standards for substances it has listed, EPA must first propose such standards for public comment and then hold a formal hearing on them. Standards for the nine substances, PCB's included, were in fact proposed by EPA on December 27, 1973. The required hearing began a month later and continued through May, 1974. Under Section 307(a) EPA is mandated to promulgate final standards not later than 6 months after publication of proposed standards, or "immediately" if EPA chooses to modify the proposed standards as a result of the hearings (ref. 60). Yet, despite this requirement, 2 years have elapsed without the promulgation by EPA of a single standard under Section 307(a).

This failure to develop final standards is being challenged in a law suit brought in the District of Columbia (ref. 63). The NRDC and the Environmental Defense Fund (EDF), plaintiffs in the suit, are claiming that the EPA's omission is contrary to FWPCA's requirements and are seeking a ruling establishing court-ordered deadlines for final EPA action on toxic pollutant effluent standards. In affidavits filed in this law suit, EPA has stated that the record of the original hearing "could not justify or support the promulgation of either the originally proposed standards or any specific modification thereof" (ref. 64). In support of this conclusion, EPA claims that the evidence at the hearing was inadequate to permit the agency to determine "what level of each of the toxic pollutants would provide an ample margin of safety as required by Section 307(a) of the Act." EPA also states that it was unable at the hearings "to either assess or respond to" industry evidence "tending to show" that the proposed standards would have substantial economic repercussions on the regulated industries (ref. 64). Accordingly, EPA now intends to repropose, i.e., to propose new toxic pollutant effluent standards, and to begin the hearing process again. The agency states that under its new schedule it "expects" to propose new standards for PCB's in late February, 1976 (ref. 65). EPA has also informed the parties to the law suit that these new PCB standards, unlike the original standards, will be limited to dischargers who manufacture PCB's or use PCB's in castings, electrical transformers and capacitors.

Behind this record of delay lies a series of complex problems which deserve to be examined in a far more comprehensive manner than we can undertake here. Our own judgment, based on our work in this area over the past 3 years, is that several underlying factors are principally responsible for this failure of Federal regulation. We offer them for further examination and discussion.

First, EPA has not had during this period a strong institutional commitment to doing something about the problem of toxic water pollutants. Partly this is a reflection of the historic concerns of the sanitary engineering profession, concerns which still dominate much of EPA's thinking, and partly it results from the failure of EPA leadership to assign priority attention to this problem during the formative period of the agency's programs. Even as Congress was developing the act, EPA informed the House Public Works Committee that "we do not endorse the provisions of Section 307 relating to toxic substances" (ref. 66). Subsequently, the resources EPA allocated to the toxic pollutant problem were miniscule in comparison with the need, and this pattern continued despite the fact that it was repeatedly criticized. As late as March, 1975, EPA's priorities in the water pollution area completely neglected control of toxic pollutants. In a discussion of priorities for fiscal year 1976, EPA failed to mention toxic pollutants in a table listing the agency's first, second, *and even third* priorities for the coming year (ref. 67).

A second factor has been the very large and well financed efforts by the dischargers of toxic pollutants to oppose development of standards under Section 307(a). Approximately 35 major corporations and industry groups challenged EPA's proposed standards in the formal hearing that was held in early 1974. The talents of many of the country's best paid lawyers and scientists were at the disposal of the PCB, mercury, and cyanide dischargers, and it seems almost certain that far more resources were spent picking apart EPA's proposed standards than EPA had used in developing them.

One theme developed by the dischargers at these hearings was the potential economic impact of meeting the proposed standards. Evidence was introduced at the hearings to show that the technology to meet some of the standards was not available or that where it was available it could only be installed at substantial expense. It was also contended that the proposal would force segments of major industries to shut down. EPA, though it has never rigorously investigated these claims, has nevertheless acted on them to the point that concern over economic dislocations has become a major inhibiting factor within the agency, overshadowing any concern with public health and environmental quality. Environmental organizations have urged EPA, unsuccessfully, not to delay standards development on these grounds, pointing out the self-interest in such claims of hardship and also EPA's obligation to implement the act as Congress wrote it. If a discharger believes that a variance from a Section 307(a) standard is justified in light of economic hardship, that plea, environmental groups have urged, should first be addressed to Congress, since Section 307(a) does not now contain a variance provision. But the absence of such a variance provision should not be used to justify inaction, or inadequate action, by EPA.

Because of recent publicity in the media of PCB-contamination in the Great Lakes region and the Hudson River, it is encouraging to note that several States have taken administrative and legislative initiatives to control the discharge of PCB's into lakes and rivers. Earlier this year, the Michigan Natural Resources Commission endorsed the recommendation of the Lake Michigan Toxic Substances Committee, calling for a ban on PCB imports and use in the United States and for a rapid replacement of PCB's in electrical capacitors and transformers (ref. 61). At the same time, a bill has been introduced in the State Legislature to place strict control on the use and sale of PCB's in the State (ref. 61).

In Wisconsin, the Division of Health of the State Department of Natural Resources has issued warnings on consumption of large size lake trout and salmon from Lake Michigan and the upper Mississippi River. The Department also held a hearing in August of this year, with a view to developing a strategy to control the sale and distribution of PCB's within the State (ref. 61). In Minnesota, an Interagency Task Force was established, principally to widen the monitoring of PCB's in fish in several rivers and streams in the State (ref. 24).

In terms of administrative remedies, the strongest action was taken this year in New York State, after it was discovered that two G E plants on the Hudson River appeared to be the major dischargers of PCB's in upper regions of the river. The New York State Department of Environmental Conservation on September 23, 1975, issued an abatement order requiring the G E plants to achieve zero discharge of PCB's by September 30, 1976. Administrative hearings begun on October 6, 1975, are expected to continue for several weeks (ref. 69). At the same time, Commissioner Ogden Reid of the department publicly cautioned consumers against eating striped bass from the Hudson River and salmon caught in Lake Ontario (ref. 70).

On the international front, the Organization for Economic Cooperation and Development (OECD) a few years ago recommended to its member countries, which includes the United States, West Germany, France, United Kingdom, Japan, Italy, and Spain, involved in the production of PCB's, to restrict the commercial use of PCB's to closed systems (ref. 71). Only Japan has taken further strict measures to completely eliminate the use of PCB's in all commercial application, including capacitors and transformers. It also prohibits the import and export of the compound, and since September, 1972, there has been a virtual elimination of almost all applications of PCB's in the country (ref. 57).

CONCLUSION AND RECOMMENDATIONS

On August 29 of this year, NRDC and the Hudson River Fisherman's Association requested the Administrator of EPA to take immediate and decisive action to curtail the discharge of PCB's into the Hudson River, pursuant to § 504 of the Federal Water Pollution Control Act (FWPCA) (ref. 72). Under § 504, the EPA is given authority to seek whatever emergency relief is needed to seek abatement with pollution sources presenting "an imminent and substantial endangerment to the health of persons or to the welfare of persons." To date, we have received no indication that the EPA is prepared to take emergency action against PCB dischargers, and with respect to the G E plants on the Hudson River, it has postponed its permit hearings pending the present State of New York proceedings against the company.

At the same time, we are very concerned about the proposal by the U.S. Army Corp of Engineers to undertake extensive maintenance dredging in shipping channels in the Hudson River. We have called upon the Corp of Engineers to hold public hearings before the agency prepares its final environmental impact statement, since in the earlier draft statement the agency had failed to recognize the problem of PCB contamination of sediments in river bottoms (ref. 73).

On behalf of three New York environmental and conservation organizations, we have intervened in the administrative proceedings now being conducted by the New York State Department of Environmental Conservation against the two G E plants in the Fort Edward-Hudson Falls region of the Hudson River. We are in general agreement with the State's abatement order of achieving zero discharge of PCB's by September next year.

Finally, we are at present, along with the Environmental Defense Fund, planning to petition the Federal Food and Drug Administration to lower the current PCB tolerance limits on foods and food products in the United States. This is, as mentioned previously, based on recent long-term data that show considerably lower "effect" levels of PCB's on experimental animals.

We recommend the following courses of action that should be taken by Federal and State agencies to address the problem of PCB's contamination of the environment:

1. A complete phase-out of the manufacture, sale, use and distribution of PCB's in this country, including its use in closed electrical systems;
2. A ban on the import and export of PCB's to and from the United States;
3. Development of a PCB use and consumption inventory, to pinpoint all point and nonpoint discharge of PCB's to the environment;
4. An accelerated program of monitoring and surveillance of PCB's in fish, wildlife, and foods by State and Federal agencies;
5. A moratorium on all dredging of river bottoms, until a complete study is conducted to examine the resuspension of PCB's in river water from such activities;
6. Promulgation by the EPA of the toxic substances effluent standard with respect to PCB's, such that water quality standards in receiving streams be no more than 0.001 ppb;
7. A significant lowering of present FDA tolerance limitation of PCB's in foods to reflect present knowledge of its long term chronic toxicity; and
8. A swift passage of the Toxic Substances Control Act, which is still pending in Congress, to provide additional authority to Federal agencies to cope with the manufacture and use of toxic substances.

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FDA REGULATION OF PCB's IN FOOD

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Abstract

Investigations by the Food and Drug Administration (FDA) during the period of 1969-1971 disclosed that PCB's had become a significant contaminant of the Nation's food supply. Spillage or leakage of PCB's from manufacturing equipment or contact with PCB-containing materials were identified as routes of direct contamination of food and animal feed. The presence of PCB's in the environment was causing the indirect, unavoidable contamination of certain types of food, particularly those of animal origin. In March 1972, the Food and Drug Administration formally proposed regulations to limit bureau exposure to PCB's from these sources of PCB's in the diet (the scientific and legal basis for these regulations, which were finalized in July 1973, are described). Presently, the problem of PCB contamination of foods, except for certain freshwater fish, has substantially diminished. Because of new scientific information on the potential hazards of PCB's on the public health and the continuing presence of high levels of PCB's in certain freshwater fish, the Food and Drug Administration has initiated a review of its temporary tolerance of 5 ppm in fish.

In 1969, the Food and Drug Administration identified the presence of PCB residues in milk from several dairy farms in West Virginia. Eventually, the source of contamination was traced to spent PCB transformer fluid that was used as a vehicle for a herbicide. The herbicide-PCB mixture was sprayed along powerline rights-of-way causing dairy cattle grazing areas to become contaminated. West Virginia officials were advised that FDA would initiate regulatory action against shipments of milk containing 5 ppm or more PCB's on a fat basis. As a result, the involved dairy farms were placed under State embargo until it was demonstrated that milk from these farms contained acceptable levels of the contaminant.

I mention this incident for several reasons. First, it represented the first documented incident in which an industrial use of PCB's directly contaminated food, in this case animal feed, which subsequently caused residues in milk. Second, it represented the first U.S. regulatory action taken because of PCB contamination of food. Third, aside from historical interests, it represented a preview and start of what was to become a complex and serious problem.

Within the next 2 years, seven other major incidents of PCB contamination of food had occurred in the United States. Not only did these so-called "industrial accidents" present a threat to human health, they also resulted in severe economic losses to the milk, egg, poultry, and feed-producing industries. Since the details on these accidents have been reported (ref. 1), I will not describe the circumstances surrounding each. Suffice it to say that by late 1971, it was quite apparent that spillage or leakage of PCB's from manufacturing equipment or contact with PCB-containing materials could, and did, directly contaminate food and feed.

During the same period, FDA and other regulatory agencies began to routinely test foods for the presence of PCB residues. It soon became evident that because of their widespread, uncontrolled industrial uses, PCB's had also become persistent and ubiquitous contaminants in the environment, causing the unavoidable, indirect contamination of certain types of foods.

Although scientific information on their hazards was limited in 1971, there was sufficient data for FDA to conclude that the presence of PCB's in food posed a potentially serious public health problem and that regulatory controls were necessary.

Accordingly, FDA developed and published a notice of proposed rulemaking in March 1972 (ref. 2) to limit human exposure to PCB's by dealing with the direct and indirect sources of PCB's in the diet. On July 6, 1973, a final order on the proposed regulations was issued in the *Federal Register* (ref. 3).

The final regulations included two main provisions: First, it banned the industrial uses of PCB's in establishments manufacturing, handling, or storing human food, animal feeds, and paper food-packaging materials. This was done to preclude the direct, accidental contamination of these articles. The ban did not apply to the use of PCB-containing electrical transformers and capacitors.

The Federal Food, Drug, and Cosmetic Act provided the statutory authority for invoking these restrictions as part of FDA regulations on Current Good Manufacturing Practices. This preventive approach means that the industrial uses of PCB's in firms subject to FDA jurisdiction would be considered a violation of the law without first having to demonstrate that products from these firms were being contaminated.

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The second provision of the FDA regulations was the establishment of temporary tolerance for unavoidable, indirect PCB contamination of milk and other dairy products, eggs, poultry, baby foods, animal feed and feed ingredients, and fish; also, since 1973, FDA has had in effect an action level for PCB's in paper food-packaging materials (table 1).

Table 1. FDA temporary tolerances for PCB's

Commodity	Temporary tolerance (ppm)
Milk	2.5 (fat basis)
Dairy products	2.5 (fat basis)
Poultry	5.0 (fat basis)
Eggs	0.5
Finished animal feeds	0.2
Animal feed components	2.0
Fish	5.0 (edible portion)
Infant and junior foods	0.2
Paper food-packaging material	10.0[a]

[a] Action level.

Prior to the 1972 issuance of the proposed PCB regulations, FDA used guidelines or informal action levels to control the marketing of foods and feeds with excessive PCB residues.

The authority to establish the tolerances for PCB's in these consumer products is found in section 406 of the Federal Food, Drug, and Cosmetic Act. This section provides that where the addition of a poisonous or deleterious substance to food cannot be avoided, regulations shall be promulgated to set limits on the quantity of the substance permitted. Section 406 contains three criteria for the setting of these limits: (1) the extent to which a limit is necessary for protection of public health, (2) the extent to which the substance cannot be avoided, and (3) the other ways in which the consumer may be affected by the same or other poisonous or deleterious substances.

In dealing with the problem of unavoidable PCB contamination of foods, FDA's principal objective was to minimize human exposure from dietary sources of PCB's. At the same time, the agency sought to avoid serious disruption of the Nation's food distribution system so as not to deprive the consumer of a significant portion of his food supply. As a result, it was considered necessary to establish separate and different limits for each type of food shown to contain unavoidable residues. Only in this way would overall dietary levels be kept at a minimum and would the public be assured that PCB's, if present in these foods, are generally within safe limits.

Thus, the selection of appropriate tolerance levels for PCB's in the different foods represented a "balancing" between potential harm to the consumer and economic impact. This balancing principle, which is mandated by section 406 of the statute, necessitated the use of subjective judgment regarding available information on the toxicity of PCB's and on the occurrence of PCB's in the food supply. In other words, while they took into account established, acceptable daily intakes, the tolerance levels were not based solely on ADI's, nor can they be explained strictly in terms of ADI's.

Data from FDA total diet studies and surveillance programs for 1969-1972 were also important considerations. These data showed that PCB residues in food were sporadic and that by eating a well-balanced diet, there was only a remote possibility of systematic exposures to PCB's at or above the ADI's over an extended period of time. As a result, FDA concluded that the tolerance levels would provide an adequate margin of safety for the public, provided dietary exposure to PCB's remained sporadic and, in time, would diminish. This approach also allowed FDA to set the tolerance levels high enough to minimize the economic impact that would result from the food and feed industries' compliance with the tolerances.

Since issuance of the FDA regulations, the agency has not encountered the types of "PCB-accidents" involving food or feed that had occurred prior to 1972. Current surveillance data form FDA, the U.S. Department of Agriculture, and others show that with the exception of freshwater fish, the presence of PCB's in those commodities subject to tolerances continue to be random and sporadic, but with an overall substantial decline in their frequency and levels.

This decrease is precisely what FDA intended when it promulgated the PCB regulations and, as I just mentioned, served as one of the basic premises for the temporary tolerances.

FDA attributes the present situation to several factors:

1. Voluntary restrictions instituted by industry with respect to the industrial uses of PCB's. This has resulted in less potential for PCB contamination of the environment and subsequent transfer to food.
2. Regulatory actions initiated by FDA, USDA, and State agencies when excessive levels of PCB's in food were encountered. This has had a salutary effect on industry practices.
3. Efforts of the food and feed industries to conform to Current Good Manufacturing Practices and to market only those products that comply with the tolerances.

I wish to emphasize, however, that the present situation does not mean that FDA considers PCB's to be no longer a problem. Since occasional shipments of food exceeding tolerance have been encountered during the past 2 years, there is still a need to monitor foods for PCB residues and to have regulations governing the presence of PCB's in the food supply. Furthermore, and certainly of a more serious nature, is the continued problem of PCB's in fish as a result of continued industrial discharges of the chemical into lakes and rivers. Needless to say, reports of PCB levels in freshwater fish from certain waters at 5 to 10 times higher than the FDA tolerance of 5 ppm are reasons for concern, particularly since there is little, if any, scientific disagreement that the regular and consistent consumption of fish at these levels may pose a potentially serious threat to the consumer.

Because of this concern, FDA recently began evaluating the adequacy of its current tolerance for PCB's in fish. This evaluation includes a review of all recently reported studies on the toxicity of PCB's, as well as a review of available data on the current PCB levels in fish and the economic impact that would result from a lowering of the 5 ppm tolerance.

If the decision is made to lower the tolerance, a proposed amendment to the existing regulation will be published in the *Federal Register* along with a detailed explanation of the basis for the proposal. Through this rulemaking procedure, public comment will be solicited and fully considered by FDA before final action is taken and implemented.

Obviously, a lowering of the tolerance by FDA will not in itself solve the many problems of pollution of the environment with PCB's. Nor will it guarantee that fish available for consumption will comply with the tolerance since only fish involved in interstate commerce are subject to FDA's jurisdiction. Thus, FDA enforcement of a lower tolerance will not necessarily result in additional protection for each and every consumer. Furthermore, even if warnings and advisories are issued by FDA and State officials, there is no assurance that sports fishermen and, perhaps others, would abide by these warnings.

The same may also be said for foods other than fish since, as long as the chemical is being used in a manner that causes its introduction into the environment, the potential for the indirect contamination of the food supply surely exists.

For these reasons, the Food and Drug Administration urges and will support the Environmental Protection Agency and State agencies in the initiation of controls aimed at the industrial uses and particularly the disposal of PCB's in order to curtail and eliminate further pollution of the environment and the contamination of foods with PCB's.

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PROGRAMS AND AUTHORITIES OF THE ENVIRONMENTAL PROTECTION AGENCY

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Abstract

The Environmental Protection Agency operates under many constraints. Its research and regulatory functions are influenced by Congress, court action, and public opinion. The time and energy required to produce even a simple regulatory action is substantial. And what appears to be a large EPA staff is actually small when the number of toxic substances and contaminant point sources to be controlled are considered. When creating regulations, EPA must attempt to make them reasonable and enforceable; the regulations then must of necessity be limited.

EPA's role with respect to the Federal Water Pollution Control Act and the Clean Air Act is herein explained. Recommendations are made that could improve the Federal/State Point Source Regulatory Program: public awareness could be increased among people who use PCB's as to their handling and disposal; there is potential for joint industry/government voluntary labelling and disposal control for PCB's; EPA could focus attention on electric utilities, the major user of PCB's; and finally, local water quality management programs funded under the Federal Pollution Control Act could be redirected so that they give PCB control greater emphasis.

The purpose of my talk is not to tell you again what either EPA has or has not done to control PCB's or to tell you what we are going to do. Quite frankly, I do not know what we are going to do beyond what you have heard explained in terms of increased monitoring, research, establishing a 307(a) toxic effluent limitation, and establishing permit limitations for known dischargers of PCB's.

Rather, what I thought I would do today is briefly to review EPA's programs and authorities. I will try to give you a feel for the context of our decisionmaking and the limitations on it so that the non-EPA people here have a better feel for what they can rely on the Federal Government to do in the future and what things they probably ought to think about doing at the State or even, perhaps more importantly, at the local level.

Perhaps the overriding concern is that Federal regulation in the environment (or anywhere else) requires a clear understanding of the consequences of regulatory action. The law requires it, the public expects it, and we are making every effort to do it. As a result, the regulatory process is not as responsive and rapid as we might like it to be Dr. Ahmed made some pointed comments about EPA progress in regulating under the Federal Water Pollution Control Act. These comments were factually correct, but I think there are probably some reasons for these things happening, generic reasons associated with Federal regulation that we should keep in mind. I do not know that there is a way, even if all the people in the Environmental Protection Agency were replaced, to change substantially the approach to environmental regulation at the Federal level.

It is important for non-Federal people to remember that the Federal Government is no more than a collection of people. The people who are going to regulate PCB's are in this room or have been in this room over this 3-day period. So when we say that EPA or the Federal Government should do something, in essence we are saying that the people sitting here, who know no more or less about PCB's than you do, should do something.

The Environmental Protection Agency is not a very large organization when you think about the scope of the problems that Congress has asked it to deal with. EPA is asked to regulate tens of thousands of stationary-point sources of air and water pollution, tens of thousands of pesticides that are being used in the environment, and tens and even hundreds of thousands of drinking water supplies. We have programs on radiation noise, solid waste, and toxic substances. In addition, we have what is considered to be the largest Federal Public Works program in our Municipal Construction Grants effort. We also have a substantial research and development program. There is no army of people to implement these programs. There are fewer than 10,000 people in the Agency with only a couple of hundred in the Regional Office here. When they are divided among the various programs and the number of sources to be controlled, and the time and energy that goes into taking even some of the most simplistic regulatory enforcement steps is considered, it seems unlikely that EPA will be able to revolutionize regulation or enforcement in the area of PCB's.

Mr. Train has made it clear that he is going to accord a high priority to PCB control, and hopefully, we are going to do substantially better than we have. However, we do not have hundreds of Federal bureaucrats to locate and control all the sources of the PCB's that are going into municipal sewers, being dumped on the ground, and running off into streams and rivers. It is important to remember that our resource allocations frequently are driven by things that we cannot control. We come under tremendous pressures from the public, the courts, and from the Congress to do or take action in various areas. A year ago, municipal construction grants received a great deal of attention—we were not spending the construction grants money fast enough or building enough municipal treatment works. With regard to regulations, our friends at NRDC have served a very valuable public service by encouraging us to overcome our bureaucratic inertia and to move faster in some areas. But in fact our efforts to accelerate the regulatory process have often resulted in the courts remanding our decisions, doubling the work, with little or no environmental benefit.

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The Federal Water Pollution Control Act, for those of you who have not had an opportunity to read it, is extremely long and complex and has a number of provisions that would bear on the PCB pollution problem. They overlap, I won't say that they conflict, but they have the potential to conflict in some cases. The act is very demanding in terms of the things which the Federal Government must consider in the process of developing its regulations. When we do not consider them, the courts require us to go back and do so.

We are caught somewhat in the middle. I see the legislative history and the nature of environmental laws, Federal Water and Pollution Control Act, and Clean Air Act as essentially having established the purpose of Federal involvement to be a reinforcement for State programs, with the State having the basic responsibility. The Federal role is to establish national standards, provide some financial and technical assistance, and to act where a State government is unable or unwilling to enforce the regulations.

Because of the way the acts are written, there are some things that EPA can do well. There are other things that the State and local governments can do much better, and there are still other things that the public can do. The strength of the environmental program is clearly with the public, and then with the State and local governments and must flow up rather than down.

Regulation, either for the control of toxic substances, of which PCB's are an example, or for the control of biochemical oxygen demand, will not be meaningful and will not have any real impact on the environment unless reasonable limitations are applied. I do not know what these reasonable limitations are for PCB's—they may be zero. The reasonableness of a regulation is determined by technology, economics, and enforceability. Based on what I have heard over the past 2½ days, there are serious questions as to the enforceability of regulatory actions which go beyond the 20 principal users.

EPA's legislative authorities and its principal areas of effectiveness focus on major point sources and discharges. With PCB's we do not have very many major point source discharges. Although at least some of those point sources seem to be creating problems, it is not clear to me, based on some of the numbers that were presented, particularly for the Los Angeles area, that controlling point sources is going to solve even a major part of this problem.

We will regulate these point sources, and hopefully we will be able to enforce those regulations so that the contribution from the major point sources will be diminished, if not eliminated. Based on quantities of PCB's in existence in the environment, we can say that those that have been produced over the last 30 years have not degraded those that have been imported. It seems somewhat questionable to me, if not unlikely, that that control of major point sources of PCB's is going to solve the fishery problem in the Great Lakes.

Let me take about 2 minutes to run through the Federal Water Pollution Control Act and the Clean Air Act, just to tell you where we are with regard to controlling PCB's. Under the Federal Water Pollution Control Act, the Federal government can establish water quality criteria, which we have done. This was done before the current act was passed. We have proposed revisions, and those final revisions have been printed and are creeping their way through the bureaucracy and toward the *Federal Register*. I expect that they will be published in final form in the next several months. This is a fairly substantial document and includes what we recommend in terms of ambient water quality, which is adequate to protect biological populations. The States in turn have to set the standards based in part on these criteria and in part on their own judgments of the local conditions.

Establishment of the standards does not solve any problems because it does not eliminate any discharges. Other sections of the act are designed to reduce discharges to achieve the standards. Section 304 provides for us to establish technology-based limitations and effluent guidelines upon which we base our industrial permits. Section 307, the toxic substance part, has turned out, at least in our minds, to be a very difficult section of the act to implement. Perhaps it will not be as difficult to implement for PCB's as it is for some of the other toxic substances and we are moving forward on it, although belatedly.

Section 307(b) calls for pretreatment and is significant because a substantial portion of the PCB's reaching our waterways are discharged from municipal treatment works and not from direct discharges on the part of industry. The pretreatment standards are going to present enforcement problems because it is difficult for municipalities and essentially impossible for the Federal Government to trace down every discharger of toxic pollutants to municipal sewer systems. We do not know whether the discharger is throwing pollutants down a manhole under the cover of darkness, whether he is discharging regularly through drainage in a building, or whether the pollutants are run off through combined sewers from a nonpoint source.

Section 311, the hazardous substances section of the act, is designed to protect from spills of hazardous substances. We have had some spills of PCB's, however, it seems clear to me that, unless someone has been hiding a lot of spills, the quantities of PCB's that are in the Great Lakes are several orders of magnitude larger than anything you could attribute to spills into the water. Spills on the land could be another question. However, once again, a few hundred Federal bureaucrats are not going to be able to chase a million capacitors or transformers and find out whether someone is releasing PCB's on the ground.

The section 402 permit program provides for effluent limitations for direct discharges to navigable waters. In addition, we have the emergency power provisions of section 504, which were alluded to by Dr. Ahmed. Quite frankly, I do not know what the criteria are for employing the emergency powers or what Mr. Train proposes to do in response to the petition.

The Clean Air Act might become a significant part of the effort to control PCB's if the numbers for the Los Angeles basin are accurate and representative. With this act, we are in a more difficult position in my opinion than we are with the Federal Water Pollution Control Act. The Clean Air Act is based on ambient standards, as opposed to technology-based effluent or emission limitations. It is unlikely that we could establish, monitor, and enforce an ambient standard for PCB's unless, by some fortuitous happening, most of the PCB's emitted into the air are coming from a few discreet point sources.

We can establish new source performance standards for air emissions, which are in fact technology standards, but I do not believe that new air emission sources are going to be the key to the PCB problem.

The hazardous air pollutant section in 112 of the Clean Air Act provides for emission limitations which are adequate to protect the public health with an adequate margin of safety. I have seen nothing in the information presented that alluded to the health effects of airborne PCB's. It seems to be more a question of the airborne PCB's washing into the rivers. I think that we would have a difficult time employing section 112.

I would like to run through what I personally think might be attractive supplements to the Federal/State Point Source Regulatory program. Based on what I have heard over the last few days, I believe that a substantially increased level of public awareness is necessary among the people who use PCB's. Working principally with labor and the management of industries using PCB's, we must try to improve the level of awareness about proper handling and disposal.

I see potential for a joint industry/government voluntary labeling and disposal control program, which would be substantially more restrictive than what we have now. Given the magnitude of the problem, it would appear that both the industry and the affected labor unions might be sympathetic in trying something along those lines to avoid the problems caused by unknowledgeable dumping of PCB's.

It seems to me that we have a major resource in our electric utilities, who apparently use the predominant share of PCB's in closed electrical systems. Electric utilities are fairly sensitive to public concern, and I would think that the electric utilities as a group or perhaps even individually might be sympathetic to mounting a significant labeling and waste-handling campaign, which they would subject to some public scrutiny.

Lastly, we are spending substantial sums of money on funding local water quality management agencies under section 208 of the Federal Pollution Control Act. It seems to me that, with what is likely to be millions of dollars available in the Great Lakes basin for water quality management, the people who live here might be well served by having the 208 agency work on the PCB problem in addition to BOD reduction, waste load allocations, and sediment laws. If in fact the PCB's remain the dominant concern in the Great Lakes basin, then maybe the 208 agency could help track down the apparent myriad of small discharge sources, to educate dischargers, and to help put controls on them.

U.S. FEDERAL AGENCY ROLES AND ACTIONS: THE DEPARTMENT OF THE INTERIOR

Nathaniel P. Reed*

Abstract

The Department of the Interior warns that its assessment of the impact of polychlorinated biphenyls upon national fish and wildlife resources, as determined by the National Pesticide Monitoring Program, reveals environmental hazards. The evaluation of data from the monitoring program has demonstrated a serious detrimental impact by PCB's upon national fish and wildlife resources and restoration programs. The Department of the Interior recommends immediate toxic substances legislation for control of domestic and imported sources of toxic substances.

During the past 2 days numerous reports have been given describing the uses and sources of PCB's, their occurrence in various components of the environment, and their real or potential threats to the health of our natural resources. The documentation of the widespread contamination of our environment by this group of compounds demands that we elevate our recognition of PCB's from a local concern to a national and global level. We must immediately respond to the dangers posed by PCB's; they represent an ubiquitous degenerative influence on our national health and well-being.

The reasons for this concern are simply stated: Our food and water supplies are contaminated; our health is threatened; and we are faced with "environmental stress" about which we know comparatively little!

The PCB problem is a national problem that the U.S. Fish and Wildlife Service has identified in both research studies and in the National Pesticide Monitoring Program. As earlier papers pointed out, PCB residues were found in all of the fish samples from 100 stations located in major river systems throughout the United States, and in almost all the bird samples taken since 1970.

I am deeply shocked by the pervasiveness of PCB's; they are literally everywhere. And I am very troubled by the exceedingly high levels found in fish from all our drainage systems; and I do not mean just the Hudson and the entire Great Lakes' system, but the Merrimac and Connecticut Rivers of the Atlantic Coast, the Mississippi, Missouri, and Ohio Rivers of the Midwest, the Columbia River system in the Northwest, the Sacramento in the West, Rio Grande River and other Gulf Coast streams, and even the Yukon in Alaska!

Fish are not alone in absorbing sizeable quantities of PCB's. The birds we are monitoring are equally susceptible. Waterfowl carry the highest residues in the Atlantic flyway, followed in descending order by the Mississippi, Pacific, and Central flyways. Further, all starlings collected since 1970 contain PCB residues.

It is one thing to trot out all these scientific facts and figures identifying quantitative residues in various terrestrial and aquatic organisms; it is quite another to assess the broad impact of PCB's upon our environment and natural resources. What are the effects of pesticides, heavy metals, and hazardous substances such as PCB's and what do they mean to our fish and wildlife habitat?

Cleansed of all the details, the facts, simply stated, are that we are in trouble. My department has spent millions of dollars, and is programming millions more in these times of fiscal austerity, on programs aimed at reinvigorating our wildlife resources, and protecting and restoring numerous threatened and endangered species.

The omnipresence of PCB residues in our environment looms as a very dark cloud casting an ominous shadow upon all of our fish and wildlife resource programs which depend on environmental quality and healthy habitats. In particular, PCB's pose an immediate threat to our efforts to preserve and protect threatened and endangered species. Under present conditions PCB's may very well pull the rug out from beneath us by destroying marginal species in spite of our best efforts for preservation.

Consider the Atlantic salmon, a species we are striving to reintroduce into our northeastern rivers. Atlantic salmon egg mortality has been associated with a threshold concentration of about 0.5 ppm of PCB. Our scientists tell me that this threshold concentration in the eggs would be equivalent to residual PCB levels in whole fish of 2.5 to 5.0 ppm, comparable to unhealthy levels found in many species throughout the United States.

I cannot justify a salmon restoration program in any northeastern river system where the fish will be subjected to PCB residues. Other recent tests show that rainbow trout eggs containing as little as 2.7 ppm of the PCB, identified as Aroclor 1242, and 90 ppb of DDT complex caused 75 percent mortality—and about 60 to 70 percent of the surviving fry were deformed.

Another test demonstrated that fish bioaccumulated more than 40,000 times the concentration of PCB in the water. I think the fish are telling us something. They are stating unequivocally that our rivers and streams are sick. We should not need a clarion call to realize that our rivers and lakes—the very lifelines of our environment—are in mortal danger.

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I am aware that it took many years of research before we could identify the effects of DDT, dieldrin, and other organochlorine pesticides in birds and mammals. We know that the reproductive success of bald eagles has been greatly reduced in Maine and along the shores of Lake Michigan where high residues of DDE, dieldrin and PCB are present in eagle eggs.

Similarly, the once abundant osprey population of Long Island Sound has suffered a near total decline as a result of chemical toxicants. Although our extensive work on PCB-fed ducks revealed little reproductive loss or eggshell thinning at environmental levels of PCB, I hope we don't have to repeatedly demonstrate the differences in susceptibility among wildlife species before we establish the imminence of a hazard requiring drastic national action. Remember that while we found quail and pheasant resistent to DDE thinning of eggshells, mallard and black ducks, american kestrels, screech owls, and brown pelicans were all highly susceptible. It pains me to remind you that the ban on DDT was not imposed until long after its threat to wildlife was proved, and action was taken only when studies showed the potential hazard to human health.

I suggest that we cease to quibble over scientific niceties and respond to the ailing resources around us. In this day and age, we should not need to be told that if the health of our natural resources is impaired, then human health is next around the corner en route to the sick bay.

We are meeting on the shore of one of a series of lakes that represents the world's greatest single freshwater resource; appropriately, they are called the Great Lakes. But how do we measure their greatness? Their immense area and volume are sufficient to characterize them as "great." They can also be considered "great" because of their esthetic and recreational value. Perhaps we could even classify them as "great" based on the amount of municipal and industrial waste they hold.

The list of attributes for these lakes is long and varied but regardless of the criteria used, the conclusion remains the same—the lakes and everything contained therein truly constitute a "great" resource.

The lakes' fisheries are particularly important both as utilizable and renewable resources and as indicators of the health of our Great Lakes. I am convinced that fish and wildlife are the true barometers of the quality of our environment. In my capacity as the steward of fish and wildlife for the Department of the Interior, and as a commissioner on the Great Lakes Fishery Commission, I have an overwhelming concern for the protection and management of Great Lakes Fishery resources.

During this century, the fisheries of the Great Lakes have suffered from heavy fishing, relentless predation by the parasitic sea lamprey, the influx of other nonnative species like the alewife, and the diminished quality of habitat as a result of waste disposal, uncontrolled shoreline development, and numerous other water polluting activities.

In recent years, however, we have taken significant steps at great financial cost to stop the decline of Great Lakes fisheries and to restore them to their former productivity. As a first step in this restoration, we undertook the control of the parasitic sea lamprey. Our cooperative efforts with the States, Provinces, and the Canadian Government achieved success in all the Great Lakes except Lake Ontario, where intensive control programs are now underway.

After 17 years and 33 million dollars, the sea lamprey, with a continued sustained control effort, no longer represents a major threat to the fisheries of the upper Great Lakes. The dissolution of the lamprey threat has allowed a successful State and Federal restocking program involving more than 150 million lake trout, splake, coho salmon, chinook salmon, atlantic salmon, and kokanee. Additional millions of rainbow, brook, and brown trout have also been stocked.

These stocked salmonids have fed heavily on the plentiful alewife and have rapidly grown to trophy size. The resulting sport fishery of these salmonids, when combined with the existing sport fisheries on other species, currently contributes approximately 350 million dollars annually to the Great Lakes States and the Province of Ontario.

All our efforts are for naught if these fish cannot be harvested—and we now face this stark possibility. A significant portion of these fish contain PCB residues in excess of the current 5.0 ppm action level established by FDA. As a result, warnings have been issued about the consumption of large lake trout and salmon from Lake Michigan. To varying degrees a similar PCB problem exists in Lakes Superior, Huron, and Ontario.

Although contamination by PCB's is particularly evident in salmonids, the problem is also present in commercial species such as carp, chubs, smelt, and alewives. Carp from Lake Michigan have already encountered marketability problems because of PCB's, and chubs appear to be at or just below the 5.0 ppm action level.

But if the current level were lowered to l or 2 ppm, then virtually all species would be restricted and essentially the entire Great Lakes' fishery would be curtailed. The current guidelines of 5.0 ppm has already tabled any serious consideration of commercially utilizing the salmonid populations in the near future. PCB's present a major problem in the future maintenance and growth of commerical fisheries. These fisheries today have a dock-side value of about $19 million annually and a total economic contribution to the United States and Canada of possibly $95 million annually when processing, shipping, and retailing are included.

We must now consider the expenditure of millions of dollars for controlling sea lamprey and stocking salmonids in the Great Lakes in the light of continued contamination of the fish stocks with unacceptable levels of PCB's. There are overriding reasons in favor of continuing our program. These include aspects of biology, economics, and ecology. The lake trout restoration program exemplifies these considerations. More than 17 years were required to restore lake trout to its present abundance in Lakes Superior and Michigan. If we stop our current programs, it is almost certain that the sea lamprey would reestablish itself and the salmonid populations and the fisheries dependent upon them would collapse, thus wiping out the past Federal investment of many years and millions of dollars and a Great Lakes economy worth several hundred million dollars. Although we know considerably more now than we did 17 years ago about lamprey control and salmonid stocking programs, it would still require at least 10 years to recontrol the lamprey and rebuild the lake trout stocks.

Why so long? The reason is a simple biological one—it takes 7 years for a lake trout to mature to an adult, spawning fish. There are few overnight solutions to natural resource restoration projects. Obviously, the quickest and most economical solution to the problem is to eliminate PCB's from the environment. With PCB's in the lakes, the entire program of restoring self-sustaining stocks of lake trout in the Great Lakes, or in fact all salmonids, is in serious jeopardy. And yet if we cannot eat these fish, I cannot justify spending millions of dollars contributed by the American taxpayer.

We cannot wish this problem away. Nothing short of immediate drastic action will enable us to raise anything better than lakes full of eunuchized fish. With the present preponderance of PCB's, the lifespan of the Great Lakes fishery will be limited to a single generation. The Great Lakes are a virtual "sink" for PCB's! It takes more than 50 years for water to turn over in Lake Michigan; there is no fast flushing action by mighty rivers or ocean energies to mitigate the lakes' absorbance of toxicants. Moreover, with more than one-third of the United States population and an even greater proportion of the Canadian population living near the lake shores, tons upon tons of PCB-infested materials are daily deposited into the Great Lakes receptacle. Transformer fluids, plastic bottles, hydraulic fluids, carbonless paper, ink, and other PCB-loaded wastes all find their way into the lakes, where the percentages of PCB's amalgamate malignantly. As soon as the food chain absorbs these wastes, the poison is off and running.

The tiny invertebrate fingernail clam has demonstrated an inordinate capacity to concentrate poisonous PCB residues and pesticides and heavy metals, which may ultimately spell the extinction of one of America's favorite waterfowl, the canvasback, which feeds voraciously on the clam. The clam was once common in the Detroit River and Lake St. Clair, habitats that used to attract major flights of canvasbacks.

The dieoff of fingernail clams on the Illinois River in 1956 spelled the end of that favorite spot for the canvasback. The canvasback represents a valuable waterfowl resource struggling against many odds as a declining species, but I foresee little succor for a population which winters and breeds in areas heavily infested with PCB's.

No dollar values can be assigned to endangered species, and even our society of technological whizzes cannot rejuvenate a species when its sole environmental niche is so totally contaminated with toxicants. The complete disappearance of Atlantic salmon from Lake Ontario during the early part of this century and blue pike from Lake Erie in the early 1960's have served as classic examples of man's impact on Great Lakes fishes and their environment. Currently we appear to be losing certain species, such as cisco and chubs, that are native inhabitants of the deepwater portions of Lakes Superior, Michigan, and Huron. Even restoration of self-sustaining populations of lake trout is in doubt in Lake Michigan because of an apparent lack of natural reproduction in the lake.

Possible reasons for these species' decline are numerous and complex. Although we have yet to pinpoint the interaction of dynamic ingredients, our scientists are certain that toxic substances such as PCB's are playing an important role. The potential effects of PCB residues in excess of 20 ppm in adult, spawning lake trout cannot be ignored.

I think I have gone far enough in detailing the problems that we face with this contaminant in the protection and management of Great Lakes fishery resources. The potential impact of PCB's on the future management of the lakes' multimillion dollar sport and commercial fisheries is tremendous, especially if the FDA lowers its guidelines, or if average PCB concentrations in fish increase.

Past attempts to control point sources of PCB's into the environment have failed to bring about any measurable reduction in the contamination of Great Lakes fishes. DDT levels in Lake Michigan fishes and mercury levels in Lake St. Clair fishes, however, have declined from between 50 and 87 percent in the past 4 to 5 years as a result of controls on their use. Why haven't PCB levels responded similarly? Do they behave so differently from DDT and mercury in the environment that we cannot reasonably expect to have observed some decline by this time? We will never know unless the flow of PCB's to the environment is stopped. I remind you that it was commonly thought that decades might elapse before DDT and mercury would decline to satisfactory levels in the great Lakes. It appears highly probable that the primary cause for the failure of PCB's to decline in Lake Michigan fish is that their sources have not been curtailed to any significant degree.

To sum up, the insidious character of PCB's is now self-evident and presents a very serious problem to all of us. These pollutants affect the very core of State and Federal fisheries restoration programs throughout the United States. Here in the Great Lakes, PCB's have the potential of severely damaging multimillion dollar sport and commercial fishing industries. We urgently need an effective program of control capable of rapidly eliminating the release of PCB's into the Great Lakes and our other national watersheds. Because of my deep concern with the problem, I support the recommendation of the Lake Michigan Toxic Substances Committee for a "national ban on all domestic and imported PCB's destined for use other than in transformers and capacitors, and [recommend] that the critical or essential use of PCB's in transformers and capacitors be immediately and critically reviewed in light of current potential replacement products."

There must be at the same time an immediate and intensive evaluation of proposed replacement products to determine their suitability for industrial use and the hazards associated with their potential loss to the environment. For the immediate range, our goal must be the elimination of all sources of PCB's in the environment within 3 years; for the long haul, however, we must vigorously pursue legislation that will preclude new "PCB's" from being discharged into the environment.

I have addressed you on my professional concerns as they apply to fish and wildlife. Now let me speak to you as a citizen. I have demonstrated that the Fish and Wildlife Service's pesticide monitoring program acts as a bellwether for measuring the future environmental impacts of toxic substances upon mankind. I reiterate that one-third of the population of the United States lives within the drainage area of the Great Lakes. The domestic water supplies for many millions of people are drawn from these Great Lakes—the same lakes whose fish populations are so contaminated with chemical toxicants that they are unfit for human consumption even under the present, outdated FDA regulations.

We are all aware that EPA is considering the establishment of PCB limits for water quality in the vicinity of 1 ppt (trillion), which is truly startling when we realize some of the lakes may already exceed 10 ppts right now. The passage of an effective toxic substances act and the complete control of PCB importation are imperative to the health of the American people. A piecemeal approach to toxic substances control would only delay and hamper our efforts to stringently regulate the occurrence of poisons in our environment.

Philosophically, we must consider man's role in his environment. Thoreau urged that we "probe the earth to see where our main roots run." The health of our fish and wildlife acts as a pulse indicator for human health. As this conference has shown, all medical and environmental gauges indicate this pulse to be unsteady and irregular. The irony of our position today is that our left hand is spending millions of dollars and thousands of man-years to establish a crucial resource while our right hand renders each new growth of this resource malignant. Our new resource will not only self-destruct but will place the human population in mortal danger.

As a final thought, let me stress that we should consider all alternatives. Since drastic action is needed, we should not be afraid to step back from industrial efficiency if we can step forward to environmental health.

Quite frankly, I am thoroughly disgusted by the gnashing of teeth, wailing, and rubbing of hands. To the agencies which have the enforcement responsibilities, a word on behalf of the bewildered but concerned American people—GET ON WITH IT!

U.S. FEDERAL AGENCY ROLES AND ACTIONS: NATIONAL INSTITUTE OF OCCUPATIONAL SAFETY AND HEALTH

Richard A. Rhoden, Ph.D.*

Abstract

NIOSH activities regarding PCB's have included: (1) the issuance of a background information document on PCB's for the occupational health community; (2) the decision to develop recommended workplace standards to be forwarded to the Department of Labor; and (3) plans for conducting retrospective mortality studies of workplaces where PCB's are processed. NIOSH is equipped to perform onsite retrospective mortality studies of workplaces, but as yet no facilities handling PCB's have requested an analysis.

To date NIOSH activity in regard to PCB's has consisted of three undertakings. On the 3rd of this month, our Office of Occupational Health Surveillance and Biometrics issued a PCB background information document. Its purpose was to inform the occupational health community of current knowledge concerning industrial uses and the toxicity of PCB's. I have a few copies of that document with me and others may be obtained by writing to our Rockville, Maryland, offices. (I might add right here that NIOSH would welcome any technical input from members of the audience with regard to case studies, hygiene surveys, workplace levels, and so forth.)

In a second action of NIOSH, it has been decided that criteria for our recommended workplace standards will be developed and forwarded to the Department of Labor Occupational Safety and Health Administration with formal development efforts to commence in March of next year. This will be an in-house effort, in contrast to the case with many of our criteria development efforts. It is anticipated that the final finished document will be transmitted to OSHA in late 1976 or early 1977. The Center for Disease Control has kindly made Dr. Kimbrough available to aid us in this effort.

At present, U.S. workplace standards for PCB's consist of environmental limits, time-weighted averages of .5 mg/m^3 for Aroclor 1254 and 1 mg/m^3 for Aroclor 1242, with skin notations. NIOSH is acutely aware of its responsibility to insure that its recommended workplace practices will not result in increased contamination of the general environment.

The NIOSH division of field studies and clinical investigations will conduct a retrospective mortality study of an as yet unselected workplace where PCB's are processed or handled. A cross sectional medical surveillance of current workers at that facility is a possibility. Such surveillance would be conducted by the medical investigations branch of that division.

To our knowledge, no such studies have yet been undertaken. A complete industrial hygiene survey will also be conducted at the selected facility, including personal air sampling, and checks of breathing zones of workers, housekeeping, and other work practice monitoring. The environmental investigations branch will conduct the personal hygiene survey.

The mortality study to which I referred will be conducted by the biometry branch. Mr. David Brown, an epidemiologist with that branch, is here and is ready and willing to discuss the protocol with interested people. The mortality study will consist of first, cohort selection—that is, determination that the selected facility has existed for at least 25 to 30 years so that sufficient time has elapsed for cancer induction and so forth. Next is cohort size determination; the cohort must be large enough to insure detection of low-incidence events. And there will be a personnel record evaluation; the personnel records must be comprehensive enough to permit followup and determination of employee work history.

Once a facility is selected for study, personnel records will be microfilmed and encoded into a data bank. The followup to determine the vital status of the cohort is then undertaken. That is, cause of death and so forth and the findings are analyzed using modified life tables in order to determine any excess mortalities and the specifics thereof.

Attempts are then made to correlate the mortality experience with workplace exposure levels, as ascertained from work history data. Section 20A7B of the Occupational Safety and Health Act of 1970 authorizes the Department of Health, Education, and Welfare to make inspections and question employers and employees as provided in section 8 of the act in order to carry out its functions and responsibilities.

The field investigations are conducted hopefully in the spirit of government, management, and labor cooperation. Section 20A6 of the Occupational Safety and Health Act of 1970 charges the Department of Health, Education, and Welfare with the responsibility for evaluating the potential toxicity of the materials used or found in the workplace. That is upon receipt of written request by employers and employee representatives.

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NIOSH is the agency which provides these onsite toxicity determinations. I found upon checking with the responsible NIOSH division that no such requests have been received to date from any facility that can be identified as a PCB facility. I have a few health hazard evaluation forms with me and I also have a few NIOSH mailing list forms and with that I'll close. Thank you.

PROPOSED CANADIAN REGULATORY MEASURES FOR PCB'S

Maurice F. Millson, Ph.D.*

Abstract

A systematic approach to the development of regulations intended to reduce environmental levels of PCB's involves three aspects: the nature of the problem, the ways of reducing the severity of the problem, and the recognition of those ways in formulating the regulations. Six regulatory packages are outlined. The Canadian legislative base on which to impose these packages is indicated, along with preparatory steps underway in anticipation of the coming into force of the Environmental Contaminants Act.

The contrast between the purposes for which DDT and PCB's have been used leads to the conclusion that presently measured PCB residue levels in the environment should not be expected to decrease, in response to Monsanto's imposed decrease in usage, at a rate comparable to that at which DDT residues have decreased.

The title given in the program is a bit optimistic. It sounds as if we made up our minds to pass information out, whereas in fact we are here to gather evidence in order to decide exactly what regulations we need. I shall deal only with an approach to regulations intended to affect the situation in the open environment, not regulations of food or other direct threats to humans. In this presentation I plan to follow a systematic objective approach to development of regulations, and it involves three aspects: the nature of the problem, the ways of reducing the severity of the problem, and how to recognize those ways in formulating practical regulations.

From the regulatory point of view, the nature of the problem is that there are dangers posed to living things by the presence of PCB's in their environment and levels do not seem to be decreasing. The dangers are a combination of the inherent toxicity of various PCB's and the exposure of susceptible species to those PCB's. Since we cannot change the toxicity of the types of PCB's, we can reduce the severity of the problem only by reducing the exposure.

Exposure incurred by PCB's in the environment is a little different from normal exposure. I think Dr. Kimbrough touched on this. For humans, one talks of exposure in terms of intake, whereas in the environment we normally talk in terms of the residues of PCB's in an organism or in a sediment. And it is those residues that we need to reduce as far as the environment itself is concerned. As a consequence, that would affect the intake of humans. We can reduce the residue levels by restricting the permitted range of uses of PCB's, by restricting losses of PCB's into the environment from any permitted uses, or by restricting the proportion of persistent homologs in the PCB's that get into the environment.

This brings us to the third aspect: how to recognize, it in the formulation of alternate regulations, the three ways of reducing the residue levels. I have put together six alternative regulatory packages of increasing severity with regard to those three ways.

The first package prohibits all uses other than in transformers and capacitors, and couples that with controls on disposal of industrial sizes of capacitors and transformers, the PCB's in them, and manufacturing wastes in making them.

The second package prohibits all uses other than transformers and capacitors. It puts a restriction on the higher chlorinated PCB's, meaning more than four chlorines, in capacitors and puts controls on disposal of industrial material.

The third package prohibits all uses other than transformers and capacitors. It restricts higher chlorinated PCB's in any capacitors, with an additional restriction on the medium chlorinated PCB's (more than three chlorines) in small capacitors because of their disposal. There are controls on disposal of industrial materials, recognizing that small capacitors are inherently unregulatable as far as disposal is concerned.

The fourth package prohibits all uses other than transformers and large capacitors, with a restriction on the higher chlorinated PCB's (more than four chlorines) in the capacitors, and has the disposal controls. This package would remove PCB's from small disposable capacitors.

The fifth package prohibits all uses other than for transformers and capacitors, and places a restriction on any PCB's with more than four chlorines. That would wipe out Aroclor 1254. It would probably not entirely wipe out the use in transformers; I think Westinghouse has always used a brand of 1242 for certain transformers.

The sixth package is total prohibition of PCB's. To any of those six there could be slight modifications. We might have to permit the use of PCB's in research; if we're going to allow them at all then we should allow their use in research, otherwise we'll never know any more about them.

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We could permit their use as a chemical intermediate. Somebody may have reason to use 4,4′-dichlorobiphenyl for conversion to something else. And with sufficient restrictions on the conversion efficiencies and the disposal of waste, it might be possible to allow them to do that. We could add an extra restriction on the concentration of the chlorinated dibenzofuran in any fluid in which it appears to be a problem or in any fluid in which it's possible to remove it or prevent it from being there in the first place.

We are currently involved in examining the evidence on which to decide what package of regulations should be recommended to those who make the final decision. How we could legally impose the chosen set? We have the traditional fields of pollution control: waste water, air pollution, solid waste disposal, and just as in the United States, in Canada there is much legislation. We have 10 provinces, all with different laws and regulations. We have the Federal Government. Between them they could cover the whole range if everyone used its authority to pass, impose, and enforce the laws that are available.

It is generally recognized, as you already heard from EPA's point of view, that those sort of laws are not adequate for the proper control of PCB's. That does not mean they are not useful in conjunction with other legislation on products.

Canada almost has a law on products—the proposed Environmental Contaminants Act. It is Bill C25 at the moment and whenever a civil servant speaks of a bill he has to be careful to say he means the proposed act, because if one talks of the act, the legislature gets rather upset that you are taking it for granted. The reason that Jim Brydon is not here today is that yesterday he had to appear at a Senate committee hearing, and my understanding from phoning back this morning is that the committee passed the bill. Now whether it passed it with one amendment I am not sure. It passed the bill, which means it will go to third reading in the Senate next Tuesday, and then all it requires is the signature of, or Royal Assent from, the Governor General or whoever is acting for him. At that point it is on the statutes books but not in force. It comes into force on a date to be fixed by proclamation. When that will be I do not know. Various people are pressing for an early proclamation; certain others feel you need time to get organized before you proclaim the act, otherwise you can get into serious problems with handling paper.

In addition to that possibility of controlling products, the provinces have constitutional power, if they care to use it, to pass laws to control in detail all industrial activities within their boundaries. They can tell industry it must do this and it may not do that. They can control them in detail. They recognize that the regulation of products is better done at the national level in order to avoid fragmenting the markets and ending up with 10 different classes of laws. The Contaminants Act implicitly recognizes provincial power and requires the Federal Government to consult the provincial governments before putting in any regulations, in fact, before publishing the proposed regulations. The consultation is to determine whether any particular province prefers to deal with the problem itself. I do not imagine that any province would want that option for PCB's, or else they would have handled it already.

How to apply the new act when it becomes available to the six packages? It contains an information gathering power, which gives us the right to demand all kinds of information if there is reason to believe a significant danger exists. Those involved with a nominated substance must identify themselves, and on request must identify what their involvement is.

The act contains three powers of prohibition. Because of the nature of its constitution base, it may only prohibit. It may not regulate in a management sense merely because we feel it better that way.

The activities that can be prohibited are those liable to injure health or the environment. With respect to something such as PCB's, this could involve prohibition of willful release into the environment and prohibition of import, manufacture, processing, sale, or use for prescribed uses. Another prohibition is that on incorporation of a substance into a product beyond a certain specified concentration or proportion.

I should point out the possible strange regulations on proportions of chlorine in PCB's. If we wish to regulate them, it would not be by naming allowable proportions of specified substances by product, but by naming classes of substances and prohibiting them for certain uses. We can name substances or classes of substances with some limitation on the type of class. These classifications are nowhere near as broad as some of those in the draft U.S. toxic substances bills.

If we combine the three powers of prohibition in the new act with the conventional pollution control powers of the federal and provincial governments, you can see that we could put in any of those six packages on PCB's. In regard to what we actually intend to do, there is no secret that the earliest regulation that would be put forward would be very simply a restriction to no PCB use other than for transformers and capacitors, with the possible exception of research and maybe chemical intermediates.

In effect that would initially confirm Monsanto's

1971 curtailment of sales. And it would not disrupt the market for electrical uses at the moment. It would disrupt whatever other markets there are that we do not have a handle on. We know some people are using PCB's. We don't know where they got them from. We have suspicions about who might be using them. Our information gathering power could be used, when in force, to find out who is using them. To go beyond the curtailment to transformers and capacitors, I cannot tell you what we shall do because we have not made up our minds. We would have to consult other government agencies or the Federal agencies. We would certainly have to discuss the action with electrical equipment manufacturers as to timing if or when we go beyond that one step.

To go to regulations on composition of fluids by prohibiting higher chlorinated PCB's, and certain chlorinated dibenzofurans, we would have to be able to specify analytical procedures. The procedure would have to be developed and would have to stand up to international comparisons; you do not do that overnight. People do not agree, for good scientific reasons, particularly on the chlorinated dibenzofurans as to exactly what are you measuring when you go through different procedures.

We have put together a good deal of information on the situation in Canada because we would have to be prepared to state the basis for a regulation. By we I mean a task force from the National Health & Welfare and Environment Departments, and the environment, health, and agriculture ministries of the province of Ontario. (Ontario controls the whole northern shore of the Great Lakes other than Michigan.) Several members of the task force are at this meeting; when we get back we shall debate our impressions of the data reported here, the opinions reported here, what we already know. We were not all of the same opinion the last time we met as to exactly what the evidence justified. We came here with open minds, but I doubt whether we shall reassemble all with one opinion. We shall be better informed, maybe a bit wiser, and a bit more scared about difficulties.

I cannot describe the Contaminants Act to you in a few minutes. It does require publication of any proposed restriction after it's been discussed with the provinces and before it comes into force. It provides for the creation of a board of review if a notice of objection is lodged. For this reason we would have to be prepared to submit to a board the evidence on which we claim to have become satisfied that the substance constitutes a significant danger to health or the environment. That is the basis of the act—the Governor-in-Council, meaning the Federal cabinet, has to be satisfied that a significant danger exists. This means that we would have to convince other departments whose interests are in promoting economic development and so on, that the danger is enough that the Federal Government as a whole should become involved. That would prevent arbitrary and capricious decisions.

We have come here partly to find evidence on the more perplexing points, in particular, the degree of persistence in the environment of the different PCB's. When they appear, one does not know for sure whether it is because they just came into that location or because they are persistent, or even which ones do persist.

On a personal level, everyone is worried that the Monsanto restrictions of 1973, '71, and '72 have not caused a significant decrease of levels of PCB's; that is, there has been no significant decrease in the levels of the persistent PCB's—1254, 1260.

I do not expect to see the persistent PCB's decrease in the 1970's in the biological accumulators, the fish and birds, and in sediments, which are nonbiological accumulators. If you look back to DDT, use dropped off from about 1963 around the Great Lakes and more or less ceased about 1968, '69, and '70. Five years later, after argument, people accepted the fact that DDT and DDE were decreasing.

All the DDT ever manufactured was intended to be spread into the environment within 1 to 2 years of its manufacture. Five years after stopping its production and use, a decrease in the environmental level has been shown. Almost all the PCB's ever made were intended to be kept somewhere because they were long-life products, and so they have not yet reached the open environment, let alone begun to show decreasing levels. One member of our task force, as a matter of fact, happens to analyze from time to time his invoices, and the carbonless copy paper invoice is still in use. It has not been manufactured for 3 years. So not only has it not yet appeared to go out of company files into the disposal system; it is not yet even in the company files—it is in the stock of unused paper.

What I do expect to decrease, if only we had the evidence, are the so-called less persistent PCB's—the 1242's or call them what you will. No one seems to have evidence or data on 1242 types in biota or sediments going back 5 years. Most people do not have any evidence for it at all, because either it is not there or they have not looked or the system cannot pick it up. There is some hope that one group in the Wildlife Service may be able to go back to some of its samples and determine the trichloro, tetrachloro levels over the last 3 years, and certainly over the next 3 years it can obtain samples and follow the trend if it exists. If that one does not decrease by 1980 or before, we have a bigger problem than anybody thinks.

PCB's IN FOODS: A LOOK AT FEDERAL GOVERNMENT RESPONSIBILITIES

Joseph Highland, Ph.D.[*]

Abstract

Our growing concern over the continued contamination of food supplies by polychlorinated biphenyls (PCB's) reflects an ever increasing knowledge of the extreme health hazards posed by these compounds. PCB's are both subacutely and chronically toxic, and have recently been shown to cause severe chronic effects at dietary intakes as low as 2.5 ppm in nonhuman primates (refs. 1-4). To date, a no-effect level for PCB's in nonhuman primates has not been established.

Federal sampling and surveillance programs have continually demonstrated the presence of PCB's in our food supply. Not only have the existing temporary tolerances for PCB's established by the FDA been insufficient to protect the public health, but recent evidence also indicates they must now be considered both obsolete and inappropriate. A new regulatory posture is needed which takes into account recent evidence of chronic effects caused by low level dietary intakes of PCB's and, more importantly, new evidence (ref. 5) substantiating the carcinogenicity of these compounds.

Such a regulatory approach is mandated by the FDA's responsibilities under the Food, Drug, and Cosmetic Act. It must involve the establishment of a zero tolerance level for PCB's in food to be achieved by means of a phased reduction to zero (using appropriately set temporary tolerances) determined by the levels of PCB's presently found in the environment. Such temporary tolerances must be sufficient to minimize the risks to public health and must be based on a level of risk which has been assessed using appropriate statistical models to account for the potential carcinogenic risk posed by any given residue level.

Appropriate regulatory action is long overdue. PCB contamination of food clearly poses a potential health hazard. The longer action is delayed, the longer society will involuntarily be required to endure such health hazards.

THE NEED TO REGULATE

The need to regulate the levels of PCB's in the food supply is brought about by our knowledge of the health hazards posed by the ingestion of these compounds.

Are PCB's Toxic?

PCB's are considered to have a low acute toxicity (ref. 6). In adult rats, the oral LD_{50} ranges from 4-10 g per kg body weight. In weaning rats, an oral LD_{50} for Aroclor[†] 1254 of 1,295 mg per kg body weight and 1,315 mg per kg for Aroclor 1269 have been reported (ref. 7). However, a low acute toxicity does not mean the absence of health hazards. On the contrary, PCB's are subacutely and chronically toxic at relatively low dose levels (ref. 8).

The chemical nature of PCB's in part may help explain why they are subacutely and chronically toxic rather than being acutely toxic. They have a low solubility in water but are highly soluble in fat (ref. 9). Therefore, they tend to accumulate and concentrate in the body's fat stores rather than being rapidly metabolized and excreted. It appears that the metabolism and excretion of PCB's, especially of those Aroclors with high chlorine content, are even slower than for some of the most persistent pesticides, such as dieldrin and DDT (ref. 6). Thus, after ingestion, PCB's persist in the body for long periods of time and thereby may elicit chronic effects.

Bioaccumulation of PCB's in Tissue

From both animal and human data we know that PCB's accumulate in adipose tissue. Animals continuously fed PCB's show a steady buildup of these compounds in their tissues; to date, no known upper limit for PCB storage has been found. Steady-state levels of dietary PCB's were reported for certain but not all tissues in rats fed 100 ppm Aroclors 1248, 1254, and 1262 for approximately 10 months (ref. 8). Once these levels were reached, they appeared to remain relatively stable for a long while even after dietary exposure to PCB's ceased. Rats fed PCB's for 58 days showed levels in their adipose tissue 5 times greater than their daily dietary intake (ref. 10). When the dietary exposure was for 240 days rather than 58 days, levels of 10 times dietary intake were noted. If PCB feeding was curtailed after 58 days and the levels in tissue measured after 71 days on a PCB-free diet, adipose tissue still showed 80 percent of the highest level previously measured while the levels in the brain showed no decrease but remained relatively constant.

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[†] The term Aroclor is Monsanto's trade name for the PCB's it produces. Monsanto is the sole U.S. producer of PCB's. The last two digits of the four-digit number indicate the percent chlorination of the product (e.g., Aroclor 1260 contains 60% chlorination). Other trade names for PCB's manufactured outside the United States include: Kanaclor (Japan), and Clophen, Flenclor, Phenoclor, and Sorol (Europe).

As noted above, significant levels of PCB's have been found in the adipose tissues of humans as well. It is estimated that levels of 1 ppm PCB or more exist in 30 to 45 percent of the general U.S. population (refs. 11,12). In a few cases, levels as high as 200 and 600 ppm have been reported. Moreover, as the PCB's continue to be consumed, the composition of the residues stored in the body no longer directly reflect what has been ingested (refs. 10,13-15). This is due to differences in the body's retention of various PCB homologs. The more highly chlorinated homologs, which tend to be more chronically toxic, are retained longer. Therefore, the toxicity of the PCB's stored in tissues may actually increase and the mobilization and metabolism of a given amount of stored PCB can produce a more severe toxic effect than the ingestion of an equivalent amount (by weight) of a commercially produced PCB mixture.

Toxicity of PCB Metabolites

It is not only the direct toxic effects of PCB's with which we must be concerned, but also the effects of their metabolites as well. Many times metabolites are more toxic than the parent compound. In fact, in the case of the PCB metabolite 5-hydroxy-2,4,3′,4′-tetrachlorobiphenyl, this has been shown to be the case (ref. 8).

The nature of PCB metabolites not only warns us of possible increased toxicological risks, but also of the types of toxicological responses that may be expected. The metabolism of PCB's by monkeys (primates like man) occurs by way of the formation of an epoxide intermediate (ref. 16). Aromatic hydrocarbons such as PCB's, metabolized via this intermediate, often produce cancer, birth defects, mutation, and cell death in experimental animals (ref. 17).

The Toxicity of PCB's in Humans

In response to PCB poisoning several adverse effects have also been observed in man. In 1968 over 1,000 Japanese experienced PCB poisoning after consuming rice oil contaminated with Kanechlor 400 (refs. 18,19). In addition to causing headaches, swelling of eyelids, temporary loss of vision, and many other symptoms, PCB's stored in the adipose tissue of pregnant women were passed through the placental wall and into their fetuses. As a result, 9 of the 10 live-born babies had unusually greyish, darkened skin, and most were born underweight. The importance of these observations cannot be underestimated, especially in light of very recent findings reported by Allen and coworkers on the effects of low-level exposure of nonhuman primates to PCB's (refs. 1-4). The effects seen in these monkey studies are similar in appearance and persistence to the symptoms of the human poisoning already discussed. Moreover, these symptoms were documented at levels of dietary intake of as low as 2.5 ppm. To date, a no-effect level for PCB's in monkeys has not been established.

Carcinogenicity of PCB's

Although carcinogenicity in primates in response to the dietary intake of PCB's has not yet been observed, it has been reported for both mice and rats (refs. 5,8). The World Health Organization has reported that Kanechlor 500 and Aroclor 1254 are carcinogenic in mice and induce both benign and malignant liver cell tumors following oral ingestion. Most recently, a team of research investigators from the Center for Disease Control, Atlanta, Georgia, the U.S. Environmental Protection Agency, the National Cancer Institute and Johns Hopkins University School of Medicine reported that 26 of 184 experimental animals (rats) fed 100 ppm of Aroclor 1260 for approximately 21 months developed hepatocellular carcinomas. Only 1 out of 173 control animals developed this lesion in the same period of time. Moreover, 146 of the experimental animals but none of the controls had tumorous lesions (neoplastic nodules) of the liver.

Appropriate regulatory action with regard to the presence of PCB's in food must take into account the following factors among others: the bioconcentration of these compounds in human tissues; the selective nature of this process which can lead to more severe toxic effects than those expected from the ingestion of the original PCB mixtures; the toxicity of the PCB metabolites; the severe chronic effects observed in nonhuman primates from dietary ingestion of low levels of PCB's; and the demonstrated carcinogenicity of PCB's in two animal species.

PCB's IN FOOD

The first evidence that food supplies had become contaminated with PCB's was reported in 1966, when fish from various Swedish waters were shown to contain PCB's (ref. 20). Since then, PCB's have been identified as contaminants in food supplies around the world. They have been found in pike taken from a lake in Finland, mollusks and marine fish from Scottish waters, a wide variety of fish caught in the St. John River system, wildlife in California, and in trout and salmon taken from Lake Michigan (refs. 8,21). More recent findings indicate that contamination by PCB's continues to be widespread. Within the last few months, striped bass caught in the Hudson River have been found to contain as much as 90 ppm PCB's, while pike caught in the Baltic Sea were shown to contain 0.31 ppm PCB in their muscle and up to 190 ppm in their ovaries (refs. 22,23). Moreover, plankton from the Baltic Sea, upon which many fish feed, contained levels of 25 ppm PCB in their fat (ref. 24).

Federal Sampling and Surveillance Programs for Chemical Residues in Food

The Federal government currently conducts three separate programs in which the contamination of foods by PCB's is measured. These programs include: the FDA Pesticide Surveillance Program, the FDA Total Diet Program, and the USDA Surveillance Program.

The FDA Pesticide Surveillance Program, which began in 1968, was developed to gather information on the levels of pesticides (including PCB's) in foods and animal feeds based on a statistical sampling program designed to cover specific geographical regions of the United States. The program is intended to assure the safety of the human food supply by screening and removing shipments of foods containing PCB's or pesticides in excess of established tolerances. Commodities covered under this testing program include dairy products, eggs, fish, animal feeds, fruits, vegetables, and processed foods.

In the FDA Total Diet Program, the levels of pesticides, PCB's, and trace heavy metals in the diet are measured. Bimonthly, a diet approximating that which would be consumed in a 2-week period by a hypothetical 15- to 20-year-old male is collected at retail stores in five regions around the United States. The foods are cooked or prepared in the appropriate manner and then divided into 12 basic food composite classes. These include meat, fish, poultry, leafy vegetables, etc. Each composite is then analyzed for contaminants.

The USDA Surveillance Program consists of periodic sampling and analysis of meats and poultry from chemical contaminants including PCB's. Samples are taken from federally inspected slaughterhouses.

The Levels of PCB's Found in Food

A summary of the findings, with respect to PCB's, of the survey and surveillance programs described above are listed in tables 1 through 3. It represents a compilation of data gathered from Federal sources (refs. 8,20,25).

Although the data are incomplete, it is clear that PCB's continue to contaminate our food. Results of the pesticide surveillance program appear to indicate an increase in the extent of contamination, while those of the USDA surveillance program indicate a decrease. This apparent conflict may be explained by the fact that different foods are sampled in these programs and it is quite possible that contamination of some foods has decreased while increasing for others.

The apparent decrease in the levels of PCB's reported in the FDA total diet survey between FY 73 and the first half of FY 74 must be viewed with caution. During the first half of FY 74, the average PCB levels for milk and other dairy products, for eggs and egg products, and for animal feed ingredients were equal to or greater than those reported for FY 73. Then why the apparent decrease? In large part this reflects an apparent decrease in the numbers of fish that were found to be contaminated with PCB's. In FY 73, 62.2 percent of the fish samples examined contained PCB's in the range of trace amounts to 123 ppm. In the first half of FY 74 only 35.3 percent of the samples checked were contaminated and the levels found ranged from trace amounts to 9.70 ppm. However, the fish data reported for FY 73 included mostly freshwater fish, where the contamination with PCB's has been shown to be the greatest, while during the first half of FY 74 the FDA samples consisted mainly of marine fish. Since the samples are not comparable, the apparent decrease observed is more probably a reflection of the difference between PCB contamination of freshwater and marine fish than an indication of a decline in PCB's in fish generally.

In addition to these observations, numerous specific incidents of PCB contamination of food have been reported by the FDA and USDA. A summary of these has been compiled by Wessel (ref. 20). A few examples follow. In 1970, the Campbell Soup Company detected high levels of PCB's (26.8 ppm on a fat basis) in chickens grown in New York State. As a result, 140,450 chickens were destroyed. In July of 1971 Monsanto Chemical Company informed the Food and Drug Administration that large amounts of fish meal might have been contaminated with PCB's during pasteurization from a leak in the heating system at their East Coast Terminal in Wilmington, North Carolina. An investigation by the FDA revealed that the leak had begun in April of 1971 and had continued through July 1971. Contamination of the fish meal was verified, and as a result, over 123,000 pounds of egg products and 88,000 chickens were destroyed. That same year, the FDA was notified that high levels of PCB's (20 ppm) had been found in Swift and Company turkeys. As a result, approximately 1 million birds were kept from market. These incidents and the results of the Federal surveillance programs demonstrate that the contamination of food by PCB's is not a new phenomenon. It is a continuing problem, which has resulted in part from the continued indiscriminate discharge of PCB's into the environment, and one which

Table 1. FDA total diet survey

Year	Number of composite food samples	Percent contaminated	Levels of contamination
68-71	900	6%	trace--0.36 ppm
FY 73	360	6%	trace--6.0 ppm
FY 74	360	4%	trace--0.05 ppm

Table 2. FDA pesticide surveillance program

Year	Number of samples	Percent contaminated	Levels of contamination
68-71	15,000	3.2%	"detectable" (fish, specifically: 1-10 ppm)
FY 73	7,882	4.0%	"detectable"
FY 74	1,723	5.4%	"detectable"

Table 3. USDA surveillance program

Year	Number of samples	Percent contaminated	Levels of contamination
71	4,175	4%	trace--15 ppm
FY 73 (poultry only)	1,037	2.6%	trace--4 ppm
FY 74 (poultry only)	574	<1%	1.25 ppm

must be eliminated because of the severe health hazards posed by these compounds.

THE ESTABLISHMENT OF TOLERANCES

Existing Tolerances

In partial recognition of the health hazards posed by PCB's, the FDA proposed the following temporary tolerances (in ppm) for the levels of PCB's which would be permitted to contaminate food:

1.	Milk (fat basis)	2.5
2.	Dairy products (fat basis)	2.5
3.	Poultry (fat basis)	5.0
4.	Eggs	0.5
5.	Complete and finished animal feeds	0.2
6.	Animal feed components	2.0
7.	Fish and shellfish (edible portion)	5.0
8.	Infant and junior food	0.2
9.	Paper food-packaging material	10.0

The FDA contends that these tolerances are sufficient to protect the public health. However, careful consideration of the available evidence indicates clearly that this conclusion is unwarranted and unsupported by the facts.

The Toxicological Basis for Calculation of The "Acceptable Dietary Intake" of PCB's

The FDA has calculated what is termed an "acceptable daily dietary intake" for PCB's. Even if the validity of such a concept is assumed, the basis for establishing such a level must be evaluated in order to determine whether the temporary tolerances that have been promulgated sufficiently limit PCB intake. In this connection the FDA's final environmental impact statement for rulemaking on PCB's (ref. 26) indicated that both animal and human toxicological data were used to establish an allowable dietary intake.

According to data avilable at that time, long-term animal studies showed no-effect levels in dogs and rats of 10 ppm for Aroclors 1242, 1254, and 1260. Employing the standard 100-to-1 safety factor, a no-effect level for man based on data derived from dogs was calculated to be 2.5 μg/kg body weight/day. From the data on rats, a 3 μg per kg body weight figure was obtained. Therefore, the allowable level of PCB ingestion in man was figured to be 0.175 mg per day for a 70-kg individual. However, according to Dr. H. Blumenthal, Acting Director, Division of Toxicology, Bureau of Foods, FDA (ref. 8), in the time since these calculations were made, there have been two important observations concerning PCB's which alter our understanding. These observations make the allowable daily intake previously calculated obsolete.

First, the recent studies cited earlier in this paper dealing with experiments on nonhuman primates were reported, indicating that the rat and dog are relatively insensitive to many of the toxic effects of PCB's. As stated previously, a no-effect level for PCB's has not yet been determined for monkeys. Second, recent work by Allen (ref. 1) indicates that the use of gross observations of toxicity are not reliable in the study of PCB's. Allen observed that routine toxicological evaluations of rats fed 100 ppm Aroclor 1248 for 1 year revealed no abnormalities in growth, mortality, and hematology. However, more sophisticated tests showed that these rates had drastically altered fat and cholesterol metabolism as well as changes in liver size and structure. Therefore, even if one were willing to accept an allowable human daily dose for PCB's calculated from animal toxicological data, one would have to question seriously the basis for the FDA's 1972 calculations.

The human toxicological data used to determine the allowable daily dose came from the accident at Yusho described earlier in this paper. The reasoning put forth by the FDA (ref. 26) was as follows:

> Since 2,000 mg was reported to be the average total dose causing an effect in the Japanese, it is possible that 200 mg total dosage PCBs (applying a safety factor of 10 to 1 as above) may be tolerated over a much more protracted period of time without overt adverse effect if daily exposure is held to minimal levels. This would permit ingestion of 4 μg per kg body weight per day in a 70-kg man. Since the lowest total dose producing an effect in man in the Japanese incident was 500 mg, a similar analysis leads to an allowable protracted ingestion of 1 μg per kg body weight per day as derived from a 70-kg man.

On the basis of these data, the FDA arrived at an allowable dose of PCB's (for a 70-kg man) of between 70-280 μg/day, although the significance of these calculations is very questionable. Particularly suspect are the estimates that had to be made of actual doses received by the Japanese, as well as the arbitrary assumption that a safety factor of 10-to-1 is sufficient to determine a safe level of intake for a "protracted period of time." In addition, the calculations fail to consider the potential long-term effects caused by stored PCB residues. Recent evidence from controlled feeding experiments of nonhuman primates which demonstrated severe chronic effects from low-level intakes of PCB's raises the question of whether similar effects might also be seen in man under the appropriate conditions.

The "Acceptable Daily Dietary Intake" Can Readily Be Exceeded

The FDA's acceptable daily PCB intake of 75-210 μg/day for a 70-kg man seems questionable at best. As

Dr. J. Wessel, Scientific Coordinator, FDA, has pointed out (ref. 20), "it is quite clear that the acceptable daily PCB intake of 75-210 μg/day could easily be exceeded by an adult consuming some of the heavily contaminated foods in his diet." Table 4 below, taken directly from the testimony of Dr. Wessel (ref. 20), illustrates this point clearly.

As Dr. Wessel concludes, "Even when PCBs are present at the tolerance level, it would be possible for an adult, with normal dietary habits, to exceed 175 μg of PCB per day." Adults with "normal dietary habits" are not the only people unprotected. Perhaps at even greater risk are those large groups within the population, such as sport fisherman or people on special diets, who consume above-average quantities of PCB-contaminated foods, or nursing babies who ingest PCB's in their mothers' contaminated milk and who, for biochemical reasons, have a very limited capacity to metabolize foreign chemicals such as PCB's. Dr. A. Kolbye, Jr., Associate Director for Science, Bureau of Foods, FDA (ref. 27), and Dr. H. Blumenthal, Acting Director, Division of Toxicology, Bureau of Foods, FDA (ref. 8), are clearly correct when they say respectively: "Infants or young children could also readily exceed the tolerable daily exposure," and "Thus, the best evidence indicates that children would be more susceptible to the poisoning caused by unmetabolized PCBs, since they have not developed effective ways of eliminating them from the body."

The Need for Reevaluation of PCB Tolerances Is Apparent

The FDA's temporary tolerances for PCB's in food are both inadequate and inappropriate. They fail to protect the populace even if one accepts the concept of an acceptable daily dose, because the dose calculated in 1972 is now obsolete in light of more recent findings. More importantly, however, the appropriateness of any "acceptable daily dose" for PCB's is highly questionable

Table 4. Relative contribution to acceptable daily intake (175 μg/g by foods subject to PCB temporary tolerances)

Type of food	Serving of food	Temporary tolerance (ppm)	Equivalent amount PCB in food	Relative contribution to ADI
Milk	800 ml (3.5 cups)	2.5 (fat basis) 0.1 (whole product)	80 μg	46%
Cheese	100 g (3.5 oz)	2.5 (fat basis) 0.6 (whole basis, assuming 25% fat)	60 μg	35%
Poultry	200 g (7 oz)	5.0 (fat basis) 0.5 (whole basis, assuming 10% fat)	100 μg	58%
Eggs	100 g (2 eggs)	0.5	50 μg	29%
Fish	200 g (7 oz)	5.0	1000 μg	580%
Packaged food	100-200 g (3.5-7.0 oz)	10 ppm (packaging)	10-60 μg[a]	6-35%

[a] Based on 1971 FDA Survey that 10 ppm PCB in packaging can result in migration of 0.1-0.6 ppm PCB to packaged food.

in light of the evidence demonstrating in experimental animals the carcinogenicity of these compounds. Since there is, at the present time, no scientific evidence that any level of intake of a carcinogen is safe, the entire concept of an acceptable daily dose for a carcinogen such as PCB is self-contradictory and scientifically unsupportable. A new regulatory posture is therefore needed.

A PROPOSAL FOR APPROPRIATE REGULATION

Responsibility Under the Law

The FDA's responsibility and obligation to protect the public health against hazardous substances in the food supply were established by Congress in the Food, Drug, and Cosmetic Act. Section 409 of the act embodies in the Delaney Amendment the explicit Congressional policy to prohibit all carcinogenic additives in food. Since "food additive" has recently been judicially interpreted to include any substance whose use can reasonably be expected to result, directly or indirectly, in its becoming a component of food,* the policy underlying the Delaney Clause is applicable to PCB's in food. In addition, Section 406 of the act authorizes the establishment of tolerances for poisonous and deleterious substances in food. An appropriate regulatory policy with respect to the contamination of food by PCB's must therefore take cognizance of these expressions of legislative intent.

A Regulatory Approach Consistent With the Health Hazards Involved

Consistent with the foregoing expressions of Congressional policy and with the evidence that PCB's are both ubiquitous in the food supply and carcinogenic in laboratory animals, the FDA should and indeed must establish a zero tolerance for the levels of PCB's permitted in food. New FDA regulations pertaining to PCB's must start with the premise that elimination of PCB's from the food supply is the ultimate goal. This does not mean that all food supplies currently contaminated with PCB's must be immediately condemned and confiscated. Such an effort would be impracticable and irresponsible. It does mean, however, that within a reasonable period of time, PCB contamination of food supplies will no longer be tolerated. In the interim, a temporary tolerance, sufficient to minimize the risks to public health, must be established, and then progressively reduced to zero. Such a regulatory posture would also require the EPA to carry out, in a reasonable and appropriate manner, its responsibilities with respect to environmental contamination by PCB's and to stop the indiscriminate discharge of PCB's into the environment.

The method used to establish temporary tolerances for PCB's is very important. PCB's have been shown to be carcinogenic in two species of laboratory animals. In light of this evidence, it is clear that the routinely applied 100-to-1 safety factor for toxic substances is completely inappropriate. The FDA has already indicated (ref. 27) its intention to adopt the Mantel-Bryan method (refs. 28,29) of statistical analysis to establish the required sensitivity of the testing method in cases where carcinogenic additives or drugs are intentionally added to animal feed or administered directly to animals. The FDA has adopted this position in an attempt to satisfy the intent of Congress as expressed in the Delaney Amendment. Temporary tolerances for PCB's, established using the Mantel-Bryan or other (ref. 30) appropriate statistical procedure, would be consistent with this policy. Any values so obtained could not be considered a "safe" level since neither the Mantel-Bryan procedure nor any similar statistical method is intended to be used to establish "safe" levels of exposure to environmental carcinogens in food. On the contrary, they are used to relate the dose of a carcinogen ingested to the probability of the induction of cancer.

The establishment of temporary tolerances must be viewed in light of the potential carcinogenic hazards posed by PCB's. The rapid reduction to zero of such tolerances is the only reasonable and responsible action that can be taken. Appropriate regulatory action is long overdue. Any additional delay in reducing dietary levels of PCB's can only result in increased exposure of the population to potentially disastrous health risks which neither they nor their elected representatives have undertaken to assume.

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CONCERNS AND RECOMMENDATIONS OF THE NATIONAL MARINE FISHERIES SERVICE REGARDING APPROACHES TO CONTROL THE POLYCHLORINATED BIPHENYLS PROBLEM

Thomas J. Billy*

Abstract

The views of the National Marine Fisheries Service concerning what can and should be done to (1) prevent PCB's from further polluting the environment, (2) protect consumers and limit human exposure, and (3) avoid unnecessary adverse publicity having a severe economic impact upon the fishing industry are described. Specific environmental, consumer, and fishing industry concerns are discussed and recommendations made in light of these concerns.

INTRODUCTION

On the occasion of this conference, the National Marine Fisheries Service (NMFS) would like to express its views concerning what can or should be done to (1) reduce the amounts of PCB's reaching the environment, (2) limit human exposure, and (3) avoid unnecessary adverse publicity.

In earlier sessions, we have heard presentations on:

1. Health effects and human exposure, including the available toxicological information.
2. The uses, sources, and identification of PCB's.
3. Discussion on the environmental fate and occurrence, including the ecological effects of exposure.
4. The availability and economics of substitutes.
5. The impact that the PCB pollution problem has already had on people and their livelihoods.

During this session of the conference, presentations have been made on what can be done to reduce the amounts of PCB's reaching the environment and to limit human exposure. Federal and State roles and responsibilities have been described and the recent Canadian regulatory measures have been presented.

With this information and data base and with the approaches and responsibilities for control outlined in this session, I would like to express our concerns and recommendations, as the Federal Agency with the primary responsibility for the management and utilization of our Nation's marine fishery resources.

AREAS OF CONCERN

Our concerns can be put into the following three categories:

1. Aquatic environment,
2. Consumers of fishery products, and
3. Fishing/seafood industry.

Let us take a brief look at each of these areas.

1. *Environmental Concerns*

Effective, comprehensive controls to prevent the pollution of the environment from the manufacture, sale, use, and disposal of PCB's in the United States have not been implemented to date, resulting in the continuing contamination of the ecosystem and the consequent occurrence of PCB residues in a number of fish species and other foods.

2. *Consumer Concerns*

We believe responsible regulatory action has been taken already to deal with the public health issue, and we fully support the current 5.0 ppm temporary tolerance for PCB's in fishery products. However, our concern is that certain information being presented at this conference and other pressures could result in precipitous further regulatory actions that are not based upon the full and complete information necessary to justify such actions.

No change in the present tolerance should be contemplated until full and proper consideration has been given to:

- a. New, conclusive toxicological information.
- b. The role of the specific species in the diet.
- c. Probable further decreases in PCB levels over an extended period of time.
- d. Direct economic impact to the fishing industry as well as the overall economic impact.
- e. Alternative government and industry approaches for assuring consumer protection.

Finally, there is concern that some of us in this room may fail to recognize our responsibility to insure that information reaching the public be completely factual, not speculative, and that it be presented fairly so as to prevent unnecessary confusion on the part of consumers and the resultant severe economic impact on the fishing industry.

3. *Seafood Industry Concerns*

There is a lack of recognition by some that an industry depends upon the natural resources which we are discussing, and we have imposed a serious problem on them that is not of their making. The seafood industry is concerned about the safety of their products and has been and continues to be willing to take responsible actions to protect public health.

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However, NMFS is concerned that the lessons from the past in over-regulating or implementing unnecessary regulatory actions will not be heeded.

RECOMMENDATIONS

In light of these concerns as well as the available information, the National Marine Fisheries Service recommends the following:

1. That Federal and State governments take immediate steps to implement effective controls to prevent the pollution of the environment by PCB's under the many authorities available to them. These controls should consider the manufacture, sale, use, and disposal of PCB's and other similar hazardous substances.
2. That current tolerance for PCB's in seafood should be maintained unless new and complete information warrants consideration of specific actions to selectively deal with specific problems.
3. That release of unnecessary or irresponsible adverse publicity or other actions that will have a direct impact on all seafood sales should be avoided.

In summary, let us not once again lay ourselves open to the criticism of having only single purpose goals. Let us evaluate all risks, costs, and benefits and then take further regulatory actions, based on the greatest net good to society.

THE ROLE OF THE COAST GUARD IN PCB POLLUTION CONTROL

Lt. Cmdr. J. A. MacDonald*

Abstract

In order for the Coast Guard to act in protecting the environment, the substance to be controlled must be designated as a harmful or a hazardous polluting substance by EPA. To this date, PCB's have not been officially established as such. Public awareness is also crucial in helping the Coast Guard fulfill its role—handlers must learn that PCB's are hazardous and treat them accordingly, and those who detect spills should report them promptly.

The Coast Guard's role in protecting the environment from PCB pollution is defined in Section 311 of the Federal Water Pollution Control Act of 1972 as amended. That role is to prevent transportation-related spills of oil and hazardous polluting substances from entering the navigable waters of the United States or the contiguous zone, and to enforce the provisions established in Section 311.

In order for the Coast Guard to initiate actions to respond to this role, PCB's must be designated by the Environmental Protection Agency as a harmful or hazardous polluting substance, and a determination made as to the amount that is hazardous. After this is determined, the Coast Guard will address the problem in three basic ways: detection of spills, prevention of spills, and enforcement of statutory sanctions for such spills.

Increased public awareness will help to reduce and mitigate the effects of PCB spills. As soon as the public is aware that PCB's are hazardous, and that special precautions should be taken in their handling and transportation, the number of spills involving PCB's will certainly decline.

Detection is exceedingly difficult because PCB's are a relatively colorless liquid. Oil, on the other hand, is more easily detected although even certain oils present problems for us from time to time. It may be possible that some form of tagging, such as the utilization of dyes, would assist in the detection of PCB spills.

Prevention also ties back into public awareness. If the stevedore handling a transformer containing PCB's on a pier in Seattle had been aware that the transformer contained a hazardous polluting substance, he might have been more cautious while handling that transformer, and the resultant spill might have been averted.

Enforcement is predicated upon an illegal spill being detected and reported to the proper authorities. We must ascertain that a spill took place, establish its cause, and identify the responsible party so that penalty assessments and statutory sanctions can be invoked.

To reduce and mitigate the effects of a spill after it has taken place, we would deal with it in a manner somewhat analagous to the manner in which we deal with oil spills. Response teams capable of dealing with spills of any number of hazardous substances of various magnitudes would assist in the containment and cleanup. Unfortunately, with PCB's these actions are made very difficult by the physical properties of the liquid.

There are some hard questions that need answering immediately. The Coast Guard, EPA, and other agencies will address them as soon as practicable. I would reemphasize, however, the fact that our future actions in regard to PCB's are predicated upon its establishment as a hazardous polluting substance.

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CONSIDERATIONS BY THE DEPARTMENT OF TRANSPORTATION

Alfred W. Grella*

Abstract

The Department of Transportation (DOT) has nine categories for hazardous materials. Packages containing these substances are to be labeled by category and handled accordingly. PCB's are not presently classified as conventional toxic materials and are therefore not regulated under DOT criteria. The department is now considering whether to develop a generic category of "hazardous wastes" and/or similar categories for environmentally hazardous materials, which would possibly include materials such as PCB's.

I have just a few remarks which would point out some of the thinking in DOT, some of the concerns we have, and some reasons why we do not have any regulatory criteria at the present time for transportation requirements for PCB's. It is important at the outset to point out that the current standards and philosophies in regulating hazardous materials in transportation are based on considerations of preventing acute hazards caused by release of hazardous materials from their packaging. This philosophy is common to both the international and the domestic transportation requirements.

Another way of saying this is that we do not regulate materials from a standpoint of the long-term effects from chronic hazards. In transportation hazardous materials are grouped or categorized into approximately nine classifications. The hazardous material may either be listed by name and classification or under a generic heading by classification.

The regulations, for the most part, provide test criteria for persons to test a material to determine its classification, if the material is not already listed. If it is regulated under these criteria, then packaging, marking, and labeling requirements are prescribed, and sometimes carrier stowage requirements are indicated.

For these purposes, the "conventional" poison classification criteria relate to the effects of animal tests against oral, inhalation, and skin-absorbtion criteria. It is clear that the PCB's that we are aware of are not presently regulated as "conventional" toxic materials. Therefore, they are not regulated in transportation under our present DOT criteria.

Previously there has never been a statutory definition of "hazardous materials." Recent legislation, as recent as January of this year, resulted in the Transportation Safety Act of 1974, Public Law 93-633. In section 103, paragraph 2, "hazardous material" is defined as "a substance or material in a quantity and form which may pose an unreasonable risk to health and safety for property when transported in commerce."

Therefore, it appears, for the first time, that we may have some sort of a basis to look at criteria other than the acute hazard, if such action appears appropriate. And the same consideration is coming up more and more when we talk about the generic term, "hazardous wastes." Many hazardous wastes may already be regulated now as hazardous materials. There are also many types of hazardous waste that may not be quite toxic enough or quite corrosive enough or quite flammable enough to be regulated. But they still may be objectionable with respect to release to the environment.

The other area is carcinogenic materials in general, which do not meet the conventional toxicity criteria.

So in these three areas—PCB's, hazardous wastes, and carcinogenic materials, we are starting to take a long look at whether there is a need for some degree of regulation in transportation. The question would then arise: What should that degree of regulation be? Certainly, packaging requirements might have to be addressed and, certainly, considerations for labeling. This will give you some idea of the things we are thinking about. We have not formed any actual, concrete plans. We are following very closely what the other agencies are doing and are maintaining as close contact as possible with those agencies.

The UN Group of Experts on Dangerous Goods, which is the international body that develops regulations for the transport of hazardous materials, has been debating the subject of PCB's and environmental pollutants for several years and has not been able to come to agreement as to whether they should undertake to regulate environmental hazards at all or whether this should be left to the environmental groups.

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OBSERVATIONS ON AND SUMMARY OF SESSION VII

Albert Kolbye, M.D., M.P.H., J.D.*

It has been a very interesting meeting and we have heard a lot of interesting data, opinions, and some rhetoric. I would like to make a few quick points that I think we should all keep in mind, because personally I feel once again we in FDA are the straight men in a bad joke. We are asked to mediate decisions that involve many parameters, not the least of which concerns human safety very directly.

We are asked to interpret data and to come up with limits on human exposure over time. We have previously dealt with environmental contaminants. We have a few that we unfortunately have experience with that are biologically persistent and for which experimental data show that there are varying degrees of carcinogenic potential, at least in animals.

I should like to talk about a guideline for a moment, because a guideline or a tolerance or however you wish to think of it represents a level in a particular food. What is really important in the long run is what the exposure dose is that humans receive and how they receive it.

The major point that I am trying to make is that we feel the average consumer is fairly well protected against the hazards of PCB's. But if one eats food with a high percentage of contaminating residues, and if one eats that particular food commodity frequently, one's exposure dose quite obviously goes up. As far as I can see, the people who in this country bear the greatest potential risk are selected sport fishermen, "sport fisher people," who, if they are catching and retaining and consuming with a high degree of frequency certain species of fish from certain geographic locations, have higher exposures than do the rest of us in this country.

One of the decisions we have to make is what incremental degree of protection will be afforded to the public by a decremental change in our guidelines. But please remember once again that a guideline itself represents a judgment concerning dietary intake, which also represents a judgment about the frequency of food consumption. And it is a little difficult sometimes to draw a bright line in a gray zone and I believe you can expect reasonable people to differ somewhat in their opinions about where that bright line should be drawn.

I would like to say one other thing about a guideline, because it would appear from time to time here and there that there has been some confusion about the meaning of a guideline with reference to cooperative action by various governmental sectors regarding the environment. An FDA guideline involving an environmental contaminant in food is not, and I repeat, is not equivalent to permitting pollution. It does not give license to pollute. Some people seem to have thought in the back of their minds that as long as fish did not exceed such and such a level of PCB's, a certain amount of PCB's entering the aquatic environment was permissible. That thinking is now obsolete.

The ecological criteria as far as I can see, with respect to wildlife, are some of the most sensitive indicators with respect to substances like PCB's, and I think, as was mentioned earlier, we should take note of such data in terms of our overall cooperative efforts to protect the environment.

I think health and environmental education should be undertaken for the public (and in particular for certain people who are concerned with disposal of solid waste) as to what containers are likely to contain PCB's and the proper identification and disposal of transformer fluid, etc.

I would also like to make one note. Let us not unduly focus on the polychlorinated biphenyls. There are other environmentally persistent compounds of concern, and a recent episode has led us to fully appreciate that the polybrominated biphenyls are also of concern. Thank you very much.

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21 November 1975

Session VIII:

SUMMARY SESSION

John L. Buckley, Ph.D.
Session Chairman

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SUMMARY OF SESSION I

DR. J. G. VOS (Rijks Instituut voor de Volksgezondheid, Bilthoven, The Netherlands): Recent findings on the biological action of PCB's, such as carcinogenicity in rodents and the disturbed reproduction in monkeys, greatly increase our concern of PCB's as a health hazard.

In summarizing the papers presented in the session on health effects, one is confronted already in Dr. Kuratsune's paper with the problem of PCB's and chlorinated dibenzofuran impurities. In the "Yusho" disease, one is struck by the persistence of symptoms present in patients, although there is a shift in the pattern, a decrease over the years in the skin lesions but a persistence of hypersecretion of Meibomian glands. In "Yusho" patients, increased urinary excretion of 17-ketosteriods was noted, as was an increase in serum triglyceride levels. Changes in the menstrual cycle were observed in a high percentage of female patients. Mean blood levels in "Yusho" patients were 7 ppb. This level conflicts with the high PCB concentrations in blood of workers who are occupationally exposed yet who, for the most part, show no indication of adverse effects.

A possible explanation for this difference was the relatively high concentration (5,000 ppm on PCB basis) of chlorinated dibenzofurans (including 2,3,7,8-TCDF) in rice oil. Apparently furans were formed in the PCB during its use as a heat exchanger, since the level of chlorinated dibenzofuran in Kanechlor KC-400 was approximately 17 ppm. Of particular significance was the relatively high concentration of chlorinated dibenzofurans versus PCB's in the liver of some patients who died, when compared with adipose tissue values. Further studies are required to solve the dilemma of the importance of furans in the etiology of "Yusho" disease; and in a wider context, to get information on possible changes in composition or concentrations of chlorinated dibenzofuran and other contaminants in used PCB's and on their fate in the environment.

During the 1971 PCB conference, there was some concern on a bladder tumor that was found in a rat by Dr. Kimbrough. Now we know that this tumor apparently developed spontaneously. But as we heard from Dr. Kimbrough, in recent studies certain PCB mixtures induce tumors in rats and mice. A spectrum of lesions are induced in livers of rats fed 100 ppm Aroclor 1260 for 21 months: hyperplastic or neoplastic nodules in 80 percent of the treated animals and in none of the controls, and hepatocellular carcinomas in 14 percent of the experimental animals and in one out of 173 controls. Metastases were not observed. However, hepatocellular carcinomas were not found in the chronic study in rats, as reported by Dr. Calandra. A possible explanation can be the small number of animals used in this study. Evidently, this question has to be solved. Hyperplastic nodules were seen in 25 to 50 percent of rats fed 100 ppm Aroclor 1242, 1254, or 1260. These three mixtures did not appear to be mutagenic or teratogenic. No-effect levels based on 2-year studies in rats and dogs and reproduction studies in rats were 10 ppm.

As reported by Dr. Allen, adult monkeys and in particular the females are very sensitive to PCB's. When Rhesus monkeys were fed 2.5 or 5 ppm Aroclor 1248—that is, approximately 0.1 or 0.2 mg/kg body weight/day—skin lesions consisting of acne of face and neck, edema, and hair loss developed in some females already after 2 months. All females exhibited these changes after 6 months, but males were more resistant. Disturbances in reproduction were present in females and not in males. Menstrual cycles were irregular within 4 months. The conception rate in the 5-ppm group was decreased. Because of resorptions and abortions, the birth rate was reduced and the infants born were small. Fifty percent of them died before weaning, showing typical lesions. These effects on reproduction may be due to an estrogen/progesterone imbalance. Female rhesus monkeys on a PCB diet had increased urinary levels of ketosteriods.

By comparing the reports of Drs. Kuratsune and Allen, one is struck by the similarity of lesions present in "Yusho" patients and those produced experimentally in the monkey, with the exception of the effect on serum triglycerides. Both species seem also to be equally sensitive in a quantitative way.

Dr. Matthews studied the effect of chlorination on tissue distribution and excretion of PCB isomers in rats. Each of the PCB's studied was removed rapidly from blood to liver and muscle. Redistribution to skin and adipose tissue increased with increasing chlorine concentration, whereas elimination via urine or feces was highest with the low chlorinated isomers. After 6 weeks, 85 percent of the 2,4,5,2′,4′,5′-isomer was still present in adipose tissue and skin, while the residue of 2,4,5,2′,5′-isomer was only 5 percent. Significant excretion occurs only after metabolism to polar compounds. The necessary factor could be the presence of two adjacent unsubstituted carbon atoms

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that facilitate the formation of arene oxide intermediates, as was also reported by Dr. Allen in his study with the 2,5,2′5′-isomers in the monkey.

Dr. Ecobichon reported on the influence of position and degree of chlorination of chlorobiphenyl isomers on the hepatic function in the rat. Substitution at the 4 and 4′ positions, irrespective of chlorination at other positions, enhanced the activities of endoplasmic reticulum bound enzymes 0- demethylase and aniline hydroxylase. This is probably due to their higher persistence. For enzymes less discretely localized in the liver cell, such as carboxylesterase and sulfobromophthalein -glutathion conjugating enzyme, the position of chlorine atoms appears of less importance. Purified unsubstituted biphenyl did produce enzyme induction, though enhanced effects were noted when one or more chlorine atoms were present.

As can be concluded from the report of Dr. Biocca, biological effects of symmetrical hexachlorobifenyl isomers differ not only quantitatively but also qualitatively. Such comparative studies are necessary for the ultimate goal—that is, the understanding of the biological effects produced by complex mixtures that endanger human health, mixtures that are different from those formulated commercially. Of the three isomers tested in mice, 3,4,5,3′,4′,5′-HCB was most toxic, followed by 2,4,6,2′,4′,6′-HCB and 2,4,5,2′,4′,5′-HCB, respectively. Liver weights were increased by all three isomers. Severe thymus atrophy and a decrease in α- and γ-globulins were produced only by 3,4,5,3′,4′,5′-HCB. In a study in chickens, these isomers along with 2,3,4,2′,3′,4′-HCB and 2,3,6,2′,3′,6′-HCB were given at 400 ppm in the diet for 3 weeks. Again, 3,4,5,3′,4′,5′-HCB was most toxic and was lethal even at 3 ppm. Chickens exhibited pronounced edema, thymic involution, and marked liver pathology. However, chickens that were fed 100 ppm died earlier and had only mild liver pathology. The 2,4,6,2′,4′,6′-HCB was less toxic (no deaths at 400 ppm) but caused marked liver pathology and highest liver weight increase. The toxicological effects of 3,4,5,3′,4′,5′-HCB differ qualitatively from the other HCB isomers studied, and resemble the lesions produced by 2,3,7,8-TCDF.

Dr. McKinney reported on metabolism studies of HCB isomers in the chicken using low resolution mass spectrometry. When metabolites were detected, the three basic reactions were isomerization, reductive dechlorination, and oxidation with and without chlorine loss. Siginificant metabolites were not detected in excreta of chickens fed 2,3,4,2′,3′,4′-, 3,4,5,3′,4′,5′-, or 2,3,6,2′,3′,6′-HCB. Metahydroxylation followed by parahydroxylation of the same ring are favored processes for 2,4,5,2′,4′,5′-HCB. Further oxidation could give quinone. The presence of a metabolite with a trihydroxypentachlorobiphenyl structure indicated that dechlorination can be concomitant with hydroxylation. A dibenzofuran metabolite was not detected.

In the case of 2,4,6,2′,4′,6′-HCB, several reaction types occurred: dechlorination, isomerization, and dibenzofuran formation. This isomer had lowest effect on body weight, did not give a high liver residue, but produced most severe liver pathology despite the dibenzofuran formation. In contrast, 3,4,5,3′,4′,5′-HCB accumulated most in liver and fat, was most toxic with "furan"-type effects, but metabolites were not detected in the excreta. However, one has to consider in the case of 2,4,6,2′,4′,6′-HCB and possibly 3,4,5,3′,4′,5′-HCB the possibility of covalently bound metabolites that may be of toxicological significance. A good correlation, which may have predictive value, was observed between the retention indices from gas chromatography with the adipose tissue concentration. An exception was the 2,4,6,2′,4′,6′-isomer, which had the smallest retention index but relatively high tissue accumulation. The strong ortho effect could be responsible for this discrepancy.

Dr. Moore reported on the first toxicity studies with 2,3,7,8-TCDF. Marked thymus atrophy and the presence of edema are seen in chickens dosed daily with 1 μg/kg. Only mild liver pathology was present at the 5-μg dose level, which produced 100 percent mortality. The LD_{50} in guinea pigs after a single oral dose was approximately 7 μg/kg body weight. In chicken and guinea pigs, 2,3,7,8-TCDF is lethal at dose levels that are less than one order of magnitude higher than that of 2,3,7,8-TCDD and share a number of biological effects. Mice and rats are more resistant to TCDF. A single subcutaneous dose of 6,000 μg/kg did not reduce body weight but gave thymus atrophy and mild liver toxicity in mice. No effect was noted in rats intubated with 1,000 μg/kg body weight. Clearly, more research is needed with different chlorinated dibenzofurans isomers in order to assess their health hazards.

SUMMARY OF SESSION II

MR. DAVID GARRETT (Environmental Protection Agency, Washington, D.C.): In Session II, Dr. James Mieure, who is research group leader, Monsanto Industrial Chemicals of St. Louis, spoke of PCB's, their properties and mixtures, and presented physical and chemical properties of commercial Aroclor products.

Dr. Stephen Safe, Associate and Professor, Department of Chemistry, University of Guelph, Ontario, presented an overview of analytical identification and spectroscopic properties. Key among the points stressed were that sophisticated methods and equipment are available for identification and quantification of PCB's at extremely low levels.

Dr. Robert Durfee, Vice President of Versar, Inc., presented topical information on the manufacture and uses of Aroclors in the United States and also gave a vivid description of the use of Aroclors in the manufacture of closed electrical systems—in particular, capacitors and transformer products. In addition he presented an overview of PCB's uses in investment casting waxes and the casting process itself was reviewed.

Thomas E. Kopp of the Office of Toxic Substances in the Environmental Protection Agency reviewed past, present, and possible future regulatory activities of EPA concerning PCB's in the water environment. He discussed proposed voluntary standards being prepared by NEMA, ANSI concerning labeling and safeguards for handling askarels and askarel-containing equipment.

Then, Mr. Stanton Kleinert, who is Chief of Surveillance for the Wisconsin Department of Natural Resources, discussed sources of PCB's in the State of Wisconsin, emphasizing the probable role of intermedia transfer from air to water and describing some possible sources of contamination for food and water.

And lastly in this section, Mr. John Hesse, Supervisor of the Toxic Material Unit, Department of Natural Resources in Michigan, described uses of PCB's and losses to the environment of Michigan. Mr. Hesse also emphasized PCB losses to air and the probably significant transfer of these PCB's into the water medium. Data was presented on PCB concentrations in municipal treatment effluents and sludges, which undoubtedly contributes to the continuous cycling in the environment of these persistent pollutants.

Some of the salient points generally brought forth in the meeting either presented or implied were that PCB losses to air could be an important contributor to the problems we encounter in water. Disposal of PCB's waste to municipal sewage treatment does not necessarily curtail the cycling and environmental damage from PCB's. And undoubtedly there should be a greater sense of accountability among users of PCB's and a closer control of inventory.

Additionally, there is presently no corrective U.S. authority which could regulate importation of PCB's and PCB-containing products, nor other serious environmental contaminants which are not pesticides, drugs, and the like.

And lastly, judging from estimates of PCB's already contaminating the environment and its inhabitants, the task of cleanup is monumental, even if we had proven technology with which to accomplish this task. Thank you.

SUMMARY OF SESSION III

DR. IAN C. T. NISBET: Eleven major points came out in the course of our session on transport and accumulation of PCB's in the environment.

1. We know almost nothing about chlorinated dibenzofurans, except that they are present in Aroclor 1242 and 1254, and we learned during Session I that they are formed in use and they are formed by metabolism. We know nothing whatsoever about their subsequent behavior in the environment.

2. PCB's themselves remain universally distributed in the environment. Although some releases have been curtailed, other releases continue. These include releases from manufacturing, leaks from supposedly closed systems, scrapping of PCB's manufactured before 1971 and of materials containing them, and use in nonclosed systems of material that

is either imported or diverted from other uses.

3. Surprisingly large quantities of PCB's which match Aroclor 1254 or 1260 are still being found in air and in dry fallout and precipitation. It is difficult to account for these quantities in terms of known past uses, and accordingly we may have to look for a significant current source of air emissions. Perhaps this may be air emissions from transformers, or disposal of scrap materials from them.

4. There is circumstantial evidence that PCB's that are now trapped in large quantities in sediments in lakes, rivers, and estuaries will remain available for resuspension and will continue to move slowly downstream. We do not have any precise information about their persistence or about the time it will take them to move downstream into the sea, but we believe that it is to be measured in years, if not decades.

5. Although PCB levels have decreased considerably in some components of the environment such as terrestrial birds or the mussels collected off California, as yet there is no clear indication of a consistent decline in PCB residues in fish. We should not expect a rapid decline, because of the long retention time of PCB's in the environment and in human tissue.

6. In addition, many items containing PCB's have service lives of 10 to 20 years before they are discarded, so that we are still experiencing releases of materials manufactured before 1971.

7. Because of time lags in response we may not yet have reached peak levels in some compartments in the environment. I am thinking here particularly of estuaries, where levels of PCB's may continue to increase as sediments are transported downstream.

8. Most human exposure to PCB's in the diet is via fish, although there is some human exposure via other routes, such as milk, meat, and by inhalation in the air. The information from the FDA total diet program suggested that the dietary exposure of an average adult in the United States is of the order of 10 micrograms per day. However, an average for PCB's has little meaning because of the very wide variations in individual consumption of fish and enormous variations in the contamination levels.

9. Individuals who have a dietary preference for freshwater fish will take in much more than the average—in some cases at least a hundred times as much as the average. Breast-fed infants appear to have extremely high dietary intakes of PCB's, averaging about 50 times higher than that of their mothers on a milligram per kilogram basis. The breast-fed infant appears to be one of Dr. Kolbye's special consumers who is imprudent enough to eat the same diet every day.

10. Monitoring of PCB residues in human fat in the United States and Canada shows that the median PCB concentration is of the order of 1 ppm. Again there are enormous variations, at least a hundredfold variation between the highest level recorded and lowest level detectable.

11. Finally, although tetrachlorobiphenyls are more easily degraded in the environment than pentachloro- or higher chlorinated compounds, the tetrachlorobiphenyls are nevertheless accumulated and retained by fish. Accordingly there are significant human intakes of tetrachlorobiphenyls, even though they are not retained in significant quantitities in human tissues. Therefore, release of either Aroclor 1016 or 1242 into aquatic systems will lead to some human exposure to tetrachlorobiphenyls.

SUMMARY OF SESSION IV

DR. DONALD I. MOUNT (Environmental Protection Agency, Duluth, Minnesota): Among and between the birds, mammals, and aquatic organisms there are marked differences in sensitivity to PCB's. Even so, these differences are not large when compared to species differences found for other pollutants. Likewise, there are differences in the toxicity of various Aroclors and isomers of PCB's that are real but not large.

While increasing chlorine content increases toxicity in warm-blooded animals, increasing chlorine content decreases toxicity of PCB's to aquatic animals. This observation is exceedingly important in determining the future actions to be taken on PCB's. Increased chlorine content seems to increase biological half-life and bioconcentration in all animal groups, but the evidence is not entirely clear cut on this point.

To generalize, we can say that in most cases acute lethal toxicity in birds and mammals occurs from 10 to 100 ppm in the food, while more sensitive species may suffer death from 1 to 10 ppm in the food. Concentrations of about 5 ppm in bird eggs produced death. And concentrations of PCB's from .5 to 5 ppm in food produced reproductive or growth effects. For aquatic animals, water concentrations of 10 to 100 micrograms per liter are acutely lethal. Concentrations of 1 to 10 micrograms per liter produced chronic reproductive or growth effects and water concentrations of 1 to 10 nanograms per liter (ppt) produced residues of biological or public health significance.

Direct uptake from water into aquatic organisms is very significant and appears to produce concentration factors on the order of one hundred thousand or more times. Water uptake supplemented by food intake results in concentration factors of five hundred thousand or more times.

The metabolic pathways, biological half-life, and selected isomer concentrations appear to be different, especially between cold-blooded and warm-blooded animals and for various mixtures and isomers of PCB's. Enzyme systems of poikilotherms are vastly different among the various groups and are also different from the enzyme systems of the homeotherms.

Measurable harmful effects of PCB's in the environment on organisms are not abundantly documented, and for the most part the effects that have been found are limited to subtle ones except for specific instances such as in mink and certain bird populations. Based upon laboratory toxicity data and contamination levels found in the environment, one would predict only subtle effects to occur. This is not to say they are not significant.

The greatest concern continues to be residues as they affect the organisms carrying that residue—especially in the eggs and sex products—and as they effect the higher consumers. Apparently water concentrations will have to be kept in the range of .1 to .5 nanograms per liter if unacceptable residues are to be avoided. And the evidence to date is unconvincing that 1016 is an acceptable substitute as far as aquatic organisms are concerned.

SUMMARY OF SESSION V

DR. NICHOLAS A. ASHFORD (Massachusetts Institute of Technology, Cambridge, Massachusetts): While the effects of PCB's and their possible control are being pursued it is quite necessary and natural that development of substitutes and their economic and health implications also be examined. In what follows, my purpose is to summarize the conference section dealing with economics and substitutes for PCB's. I shall present the summary in an order different from that in which the participants appeared, so as to follow a logical sequence most meaningful to the substitute issue. Duncan McArthur from the Foster Snell operation presented an analysis of the PCB case which was part of a larger study (called "the Snell Report") of the probable effects of a Toxic Substance Control Act. In the PCB analysis, they addressed the direct and secondary impact of a complete banning of PCB's in the absence of technological breakthroughs and new substitutes. They postulated this prohibition would be effected over a period of 76 months, 38 months of which would be consumed with hearings and the promulgation of standards, with 38 months remaining for the technological response to eliminate the use of PCB's. The analysis focused on transformer and capacitor manufacturing having a volume capacity of 45 million pounds in 1973. On this basis, they calculate a one-time cost to society of $13.7 million, $8.8 million of which would affect the secondary users. Thereafter, an annual cost of $110 million per year was anticipated, of which $16.5 million fell upon primary users and $93.3 million on the secondary. There was no detailed examination of the health and economic benefits to be derived from a banning of PCB's.

Dr. Dale Hattis from MIT's Center for Policy Alternatives reported on an examination of both economic and environmental health changes that may have occurred as a result of the past partial withdrawal of the product from commerce by Monsanto. One of the factors covered was the increase in fire insurance premiums, estimated as possibly in the tens of millions of dollars and which presumably represented the increased risk as viewed by the insurance industry. It is important to note that the methodology was restricted by the availability of information on the nature of substitutes. This points out the complication of trade secrecy and proprietary information, which, while presumably legitimate for other reasons, does complicate the assessment of costs and benefits.

Two surprises appeared in the study. One had to do with the use of PCB's in paint systems. It was noted that a major group of substitutes evolved which were more economical than the PCB's which they replaced. This evidences a common inertial resistance to technological change even when it could be anticipated to be more beneficial (in a strictly economic sense) than technology already in use. A secondary benefit which should result from the decreased use of PCB's is a reduction in the threat to sport fishing. A calculation of recreational value could be very large even compared with the alternative recreational activities which occur as second best choices on the part of sport fishermen. The enormous size of the valuation inferred for sport fishing makes it easily the largest of the defined costs of unrestricted PCB disposal.

A third participant, Richard Rollins, spoke for the Electronic Industries Association, which is concerned for the manufacturers and users of capacitors. He cited the issues of flammability and reliability, and he urged the development of less toxic substitutes. This latter remark was consistent with one very clear message embodied in remarks by the group, namely that his industry was much more concerned with the environmental consequences than cost increases in the product. He stated that there are no commercially available substitutes in AC capacitors, including the substitutes presented at this meeting, which are acceptable for safety and reliability. One interesting point made was that since the components of capacitor-grade PCB (Aroclor 1016) are not what are found in the environment, protection of the environment might not profitably be served by attacking the issue of capacitor use and disposal. It appeared to him that PCB contamination must be coming primarily from some other industrial source.

Dr. E. J. Inchalik for Exxon Chemical reported on capacitor substitutes based on diisononylphthalates with a flashpoint of 430° Fahrenheit and which are essentially already developed, but not yet fully proved and tested for health and environmental effects.

The Prodelec operation from France presented an approach which is an incremental modification of previous technology. For transformer use, they recommend keeping trichlorobiphenyl in mixtures with chlorobenzene. Both compounds of course, being chlorinated, have problems in that regard. Secondly, for capacitors they propose a mixture of dichlorobiphenyls and their alkylated derivatives, a commercial mixture of which is called chloralkylene. These products are now being examined by an institute in Bonn for environmental behavior and he indicates that tests already show there is more biodegradation and lower toxicity for these compounds than for their PCB counterparts.

Mr. David Wood from Monsanto Company described two new products, MCS-1238 and MCS-1588, for capacitor usage. He warns that neither is fire resistant although they have much better environmental characteristics. Their degradation is more rapid and their accumulation potential in the environment is considerably reduced. Occupational and environmental health considerations indicate a better product with lower acute toxicity. He also announced that they do have candidates with intermediate fire resistance between PCB's and mineral oil, but did not elaborate on this further.

Dr. Dean Branson from Dow Chemical Company announced a substitute for use in power capacitors. This compound is called XFS-4169L and is a butylated monochlorodiphenyl oxide. Once again we find that a substitute is like the original, a chlorinated compound. Its properties are worth noting however. Dielectric losses are lower or slightly lower than for capacitor-grade PCB, while the discharge inception voltage is significantly higher. The size or volume per unit of high-voltage power factor correction, called kVAR, is about that of the PCB counterpart, which means that no redesign is necessary for capacitors. There is little fire and explosion risk indicated by comparing the flashpoints and firepoints of PCB and XFS. The flashpoint for PCB is 166° centigrade, for XFS 174°. The firepoint for PCB is greater than 316° centigrade, while XFS is 199°. This substitute is reported to be more reliable and is anticipated to have a reasonable long-term price. The increased expense is estimated to be less than $20 for a 200 kVAR unit. The capacity in the first quarter of 1976 should be in the range of 1 million pounds per year, and a multimillion pound per year volume capability is estimated by the end of 1976. This substitute is more biodegradable by a factor of 45 and the bioconcentration is 30 times less. There is little toxic activity in acute and in 90-day tests, although longer-term tests need to be carried out. The accumulation in fat is 22 times less. The toxicity in fish is on the order of 20 times less than for PCB's.

carried out. The accumulation in fat is 22 times less. The toxicity in fish is on the order of 20 times less than for PCB's.

Perhaps the most exciting substitute for use in transformers was announced by Dow-Corning (represented by Richard Montgomery). He indicated that the global silicone industry had been looking for a replacement for the transformer fluid PCB's for 5 years, and the technology was now developed for the new fluid. It was to be commercially available in 1976 and had no known environmental problems. Commercial production was available to handle the global market. In the United States there has been field-testing for 4 years and the product is now ready to be used. The product is called Q2- 10 90 dielectric fluid, and is a dimethyl silicone which has been used in the military, in Japan, and in a small experiment in Midland, Michigan. He pointed out that this was not very dissimilar to "di-gel," which is used of course for human consumption. It is less flammable than many PCB's with low heat of combustion, high flashpoints and firepoints, and the product is self-extinguishing in case of ignition by violent transformer breakdown. Insurance companies have OK'd the substance for use indoors. The cost is higher than PCB's but "not outrageous," giving rise to an estimated cost increase of about 6 percent in the transformer equipment. Of course, the fluid itself is more expensive, with a higher percentage increase in cost. PCB transformers now in service can be retrofitted and he believes that the economics are favorable enough to stimulate competition. It is worthy to note that unlike the trade industries representing the capacitor users presented at the conference, there was no transformer user or producer represented. It would be interesting to see what General Electric's viewpoint would be on the potential use for this transformer fluid substitute since they have the capability to manufacture silicone fluids. Their absence at the meeting might leave one with an unwarranted optimism for the use of these fluids in transformers. One further reflection is that we are badly in need of a frank assessment of flammability risk requirements since the substitute products vary in their flammability over the great range between mineral oil and PCB's. It is not clear what tradeoffs should be made between the fire risks and the safety risks in the industrial setting and the possible damage to humans from substitute products which might get into the environment. It is also not clear that PCB's flammability characteristics should remain the benchmark for compliance although the insurance industries may see this differently.

SUMMARY OF SESSION VI

MR. CHRISTOPHER M. TIMM (Environmental Protection Agency, Region V, Chicago, Illinois): From a very broad mixture of topics and points of views that we were listening to last night, I believe the following four points should be emphasized.

First, on top of all the human health and wildlife effects that have been documented and thoroughly discussed, PCB's are having a definite and drastic effect on the livelihood and future of the freshwater fishery and fishermen. This is a very real problem to the people in various parts of the continent and they see no improvement in the near future.

Second, a ban on PCB's and better control of other toxic substances to protect our natural resources is long overdue. However, the actions of the regulatory agencies such as EPA and FDA must be based on scientific fact in the overall impact on the nation.

Third, universally there is a belief that the governmental bodies and agencies for whatever reason are far, far too slow in solving environmental problems like PCB's. This is a frustration to everybody.

And finally, there is a need to find some way to compensate the people like fishermen whose livelihood is impacted by environmental pollution which is PCB's. They do not cause the problem, they cannot control the problem, and they often cannot find anyone to blame or even talk to about it. But they suffer the consequences of decades of incomplete evaluations and testing of new compounds. Thank you.

SUMMARY OF SESSION VII

MR. CHARLES N. GREGG, JR. (Environmental Protection Agency, Washington, D.C.): Because most of you have heard the discussion this morning, I will try not to repeat it, but to provide some insights and perhaps some of the highlights. I will not try to give equal time to the various speakers.

First, there is an obvious distinction between physical and technical methods of control, which have been discussed in other sessions, and the regulatory and voluntary actions for achieving those controls that were discussed by this morning's panel.

Second, there is a distinction between the direct control of human exposures as, for instance, through limitations on PCB's in fish that can be sold for human consumption, and limitations on use or limitations on emissions into the environment, which in the long run contribute to diminished exposure. In our present situation, as you were told by the speakers this morning, there are a vast number of Federal agencies with roles. Our Government and the Canadian Government both contribute to solutions of the Great Lakes problem. We have a lot of State Governments with roles to play. One of the major problems will be sorting out roles, and finding adequate cooperation in performing those roles, among the several agencies and units concerned.

A further complication is that both in Canada and here we not only have a number of regulatory authorities now, but in each case we are looking for additional authority which it is anticipated will permit much more finely applied solutions. And at the same time that we plan for the future, we do not know for certain what authority we will have available for use.

One observation I made was that there seemed to be very little real optimism. If we applied all our available resources and used all of our authorities as best we could, we could solve in any short time frame the most pressing problem, which appears to be the high levels in fish in a number of places where they are caught and consumed in substantial quantity. This lack of optimism did not, nevertheless, suggest to the speakers that we should not take action. Everybody seemed to agree that it was appropriate to move ahead as best one could with the full range of authorities and voluntary programs available.

Also, there was considerable emphasis on nonregulatory action, and increasing communication with people who have the opportunity to affect the flow of PCB's first from industrial situations into the environment and thereafter within the environment to locations where they can lead to higher human and environmental exposures. In my view, a voluntary program seems to present a very great opportunity for improvement, perhaps much greater than a strictly regulatory program in the light of some of the difficulties experienced in using our current authorities.

Relatively less, it seemed to me, was said by this morning's speakers about just how to control PCB's, particularly how to control, through regulation, those PCB's which are currently in industrial use or elsewhere in products but not yet released generally to the environment. There was some reference to controls over disposal, but to my best recollection, there was no indication that we had any directly applicable authorities with respect to disposal.

Let me touch on a couple of this morning's highlights. Dr. Ahmed of the NRDC asserted that the government had failed to carry out its responsibilities adequately, particularly under Section 307(a) of the Federal Water Pollution Control Act. He felt that we had a schedule on which we should have produced regulations a couple of years ago, with compliance a year ago, and he criticized EPA's lack of attention and priority to toxics in our Water Pollution Control program. He made several recommendations: a phaseout of the manufacture and use of PCB's, a ban on exports and imports, development of an inventory of point sources, additional monitoring in fish and elsewhere, a moratorium on river bottom dredging until we have better indications of the trouble we stir up, effluent standards that would allow us to arrive at 1 ppt in water, lower FDA tolerance levels, and the passage of the Toxic Substances Control Act.

Mr. Wessel of the Food and Drug Administration referred to its action in 1972 and 1973 to set tolerance levels, indicating that various concerns had to be balanced in setting levels under Section 406 of the Food, Drug, and Cosmetic Act. He indicated that FDA was examining the current tolerance level in fish and considering whether or not to lower it. He said that there would be adequate opportunity for public comment on any proposal that was made. But he felt that this would not solve the PCB problem and it would not necessarily prevent consumption of fish with high levels of PCB's by private fisherman.

Dr. Kolbye of FDA added a discussion of some of the problems in setting tolerance levels, and something else I thought was significant: that steps are particularly needed to identify where PCB's are located so we can look to their proper disposal, and education to this end is needed. He did feel that the average consumer is fairly well protected against PCB's by the current tolerance level, but acknowledged that there would be higher exposures in some cases and that this was not a concern to pass over lightly.

Mr. Barber of the Environmental Protection Agency, after talking about some of our authorities and programs we have undertaken so far, gave considerable attention to some of the realities involved in EPA decisionmaking: the complexity and implications of taking various regulatory courses, and the fact that there are limited resources in EPA, particularly people, to handle a wide array of regulatory responsibilities. He pointed out the importance of the State role, and indicated that the Federal Government's role can best be viewed as one way of encouraging the State to find ways to perform a great deal of the regulatory and enforcement activity. He felt that regulation must represent some sort of consensus and be enforceable in order to be productive. He felt that a number of PCB problems were difficult to control because of the numbers of nonpoint sources.

He urged that we try for more awareness among the people who use PCB's, particularly the utilities, and suggested voluntary labeling and use of the planning process under Section 208 of the Federal Water Pollution Control Act.

Secretary Reed described both the environmental problem caused by PCB pollution and the resulting fisheries resource problem. High investment in restoring the Great Lakes fishery and future investment in this and other fisheries would be hard to justify unless we can control the PCB situation. He suggested particularly that since we find great reductions in DDT and mercury as a result of regulation, it would certainly be worth a try to see if we can reduce PCB levels in the water and in fish by taking all of the regulatory steps we now can. He endorsed the Toxic Substances Control Act, and suggested we cut off the importation of PCB's.

Dr. Rhoden outlined the plans of NIOSH.

Mr. Billy of the Marine Fisheries Service indicated their environmental, consumer, and public information concerns. They recommend not lowering the FDA level until there has been a sound attempt to understand the course to be followed, and the avoidance of unproductive and unnecessary adverse publicity.

Mr. Grella of DOT briefly described their incipient program to apply DOT authorities to PCB's, though not much has yet been done.

The Coast Guard has a number of functions under the Federal Water Pollution Control Act in the event of spills, but the Coast Guard representative told us that until we in EPA have designated PCB's as a hazardous substance, the Coast Guard is unable to go forward to use its authorities. He urged public awareness of the needs for good handling and precautions in transportation.

Dr. Millson of the Canadian Government urged a systematic approach, and outlined six regulatory control packages that Canada was considering. He emphasized that Canada does not have a use control act yet, but that such authority together with their other authorities would permit them over some period of time to do a great deal about controlling PCB's.

Since Dr. Highland spoke last, and I have exhausted my time, with apologies to him I hope you remember what he said.

CONFERENCE HIGHLIGHTS

Richard A. Carpenter*

A summary of this complex and comprehensive conference may be more useful if the information is organized in the format shown in the accompanying figure (figure 1). The presentations have dealt with three general topics:

- A. The occurrence of PCB's in the environment—sources, rates, routes, and sinks;
- B. The toxicity of PCB's—inadvertent human exposure, laboratory animal experiments, wildlife, fish, and ecosystems;
- C. The uses and benefits of PCB's to industrial society and the possibilities of control.

In each topic, information can be characterized as to:

1. What is known and generally agreed upon with a reasonable degree of confidence.
2. Remaining uncertainties and the reasons why a lack of understanding continues. Science is probabilistic in nature and this concept of uncertainty must be recognized when technical information is used in deciding issues of public policy.
3. Finally, the conference discussions have suggested some courses of action which are pru-

	A. Occurrence in the Environment	B. Toxicology	C. Uses, Benefits and Control
1. What is known			
2. Remaining Uncertainties			
3. Prudent Actions			

Figure 1. Organization of information about polychlorinated biphenyls.

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*Executive Director, Commission on Natural Resources, National Research Council, Washington, D.C.

dent, i.e., could and should be set in motion. These include setting priorities for obtaining more information and reducing uncertainties through additional research and monitoring.

The conference was not designed to produce consensus judgments or to make recommendations. Thus, the following summary must be viewed as the responsibility of the author alone.

A-1. What is known about the occurrence of PCB's in the environment

We have learned what should have been apparent from the start—that any persistent, mobile, and foreign or exotic compound is going to be detected everywhere in the environment. In addition to current production and uses, which may result in leaks, a nonpoint source situation exists in that about 400 million pounds of PCB's are already discarded in the environment and substantial amounts are apparently recirculating in sludge, sediments, in air and dust, and in contaminated organisms.

There are about 300 million pounds in industrial service and this material will be difficult to contain completely as it moves toward disposal. The sediments in lakes, rivers, and estuaries provide a very large reservoir of PCB's for contacting aquatic organisms. The material in dumps (three-fourths of the total discarded) appears to stay there for the most part. Current processing losses are important and can be decreased.

Eventual removal is to sediments in the deep ocean and we need to know more about the fate of this material in the marine environment.

It is too early to see the effects of the 1972 voluntary action to limit uses to nondispersive applications, but certainly it was a justified decision and a move in the right direction.

There will be occasional catastrophic spills which can cause substantial damage to fisheries in the future. These will be localized impacts, and will be costly to clean up.

Incineration is an effective method of destroying PCB's if the temperature is high enough (i.e., about 2,700° F).

Analytical procedures now give confidence that we can measure PCB's qualitatively and quantitatively down to a few parts per trillion.

A-2. Uncertainties about PCB's in the environment

There is a real question as to whether the PCB content of the waters of the Great Lakes (particularly Lake Michigan) can be lowered by any degree of tightening-up of the current use situation, because of the recirculating contamination that is already there. There is the question as to the role of air transport and the recirculation of material that is deposited in rain or in dust back into the air so that it is continually redistributed around the world. The composition change of PCB's in the environment with aging is not well understood; there are questions as to their chlorine content changing and regarding formation of diobenzofurans.

We need a mass balance and a model for transport and removal in order to estimate the time that will be required for these materials to eventually move to an ultimate sink in the deep ocean sediments.

A question has been raised as to the possible conversion of these materials into higher chlorinated compounds during sewage chlorination.

We need to know more about the presence of the materials in whole water; i.e., are we measuring material on suspended sediments, in very small organisms, in micelles, or actually dissolved in water? We need to know about the biological activity of these various forms of occurrence of PCB's in water.

A-3. Prudent actions

What can we do based on this information and uncertainty? We can separate, to some extent, the problem of what is already in the environment from the control of material that has not yet been released. Even if all manufacture and use were stopped immediately, environmental contamination would remain for some time to come. The PCB problem should be considered in the context of other halogenated hydrocarbons in the environment and it should not be separated from investigation and control of the residues of pesticides and other halogenated materials. Work should begin on a more systematic monitoring system and on the use of indicator organisms, such as the suggested "mussel watch," on a worldwide basis.

We can use some ingenuity in devising means of cleaning up contaminated water. This problem is of a magnitude that allows one to think about filtration or adsorption concepts for waters that are somewhat confined.

Ingenuity may suggest ways to make contaminated fish suitable for use in animal feeds or human consumption. Perhaps segregation of certain organs or particular methods of cooking would serve this purpose, or some other way of decontaminating these valuable animals might be found.

B-1. What is known about toxicity

The Yusho incident proved the toxicity of used heat transfer fluid to humans at a concentration in cooking oil of about 1,000 ppm of PCB's. But interpretation of this incident is confounded by dibenzofuran contamination of the PCB fluid. Industrial hygiene experience is somewhat reassuring as to toxicity to humans except that the occasional symptoms and illnesses cannot be related to known exposure doses in most instances. The good housekeeping practiced by some large users and the American manufacturer will probably be better than that of the variety of secondary users. No human deaths have been caused directly, but reports of serious illness and persistent skin problems continue.

Exposure of the general public can be controlled by avoiding food (mainly fish) contaminated with PCB's. Exposure from environmental contamination or direct introduction into humans is unlikely. Averages for dietary intake can be misleading since some persons may eat large quantities of fish.

Laboratory animals show effects when fed diets containing contamination levels found in fish. Fish are affected at the range of PCB levels found near industrial outfalls into ambient waters, and the occurrence of fin rot in fish near these "hot spots" is a direct confirmation of laboratory tests as to the effects on fish.

Ecosystem simplification (fewer species and lower populations) has been found in laboratory experiments to occur at concentrations similar to those in hot spots in natural ambient waters and could be expected to be found at sludge disposal sites. It is hard to find evidence of damage to terrestrial ecosystems. Biomagnification, i.e., the ratio of the PCB content in aquatic organisms to that of the water in which they live, can be as high as several hundred thousand.

B-2. Uncertainties as to toxicity

There is confusion over the interpretation by pathologists of organ damage. It would be helpful if the highly technical problems of ascertaining carcinogenicity could be worked out by those scientists involved apart from the complexity and emotionalism of a specific issue such as the PCB problem. The effect of dibenzofurans, which may be present as impurities or as metabolites, is still in question.

There is a great variation as to the response of different species in laboratory animal tests. The range of sensitivity is large. More needs to be known as to whether the reproductive problem is the most important effect. Fat metabolism is not well understood and since a great proportion of the human population is carrying around a few ppm of this material, we need to know more about it.

Wildlife toxicity data is poor and we need to know which wildlife species may react as do chickens and mink.

The relationship of toxicity to chlorine content of the PCB's is uncertain. Is the ability to metabolize these compounds (which varies with the chlorine content) desirable or not? The possibility of dioxin as a degradation product has been raised. We need to know about the equilibrium distribution of these materials between fish and water. Is it possible that depuration can be a help? Are PCB's further accumulated up the food chain after the biomagnification from water to the first organism?

B-3. Prudent actions based on toxicity knowledge

Certainly more experimental work in animals and careful replication of reported tests are necessary. Priority should be given to chronic tests in various species, detection of behavior change in animals, and laboratory work at true environmental levels, rather than to greatly increased levels of dose. Obviously it is of great importance to determine unequivically whether the PCB materials are carcinogenic.

We need to coordinate this work with experiments and observations on other halogenated hydrocarbons, particularly the chlorinated hydrocarbons, and to look at combined effects on organisms, communities, and ecosystems.

NIOSH should extract the maximum from industrial hygiene data because these human exposures do not necessitate the inferences from animal tests to human toxicology.

The widespread occurrence of PCB's in the environment constitutes an inadvertent experiment on human populations and the biosphere. A concerted effort at monitoring and analysis is indicated in order to gain the most useful knowledge for further decisions concerning toxic substances at low levels in the environment.

C-1. What is known about uses, benefits, and control problems

We know that about 700 million pounds of PCB's have been manufactured, but the current annual production volume is down to 40 million pounds from an 80 million pound level in 1970. About one quarter of present production is going into small capacitors that are scattered very widely throughout our industrial society.

The fire retardant benefits of PCB's are real and important. An abrupt change in the availability of PCB's would be disrupting, but that is unlikely in any event. The estimate of 76 months to reach an end to manufacture if the Toxic Substances Act existed today suggests that industry will have a substantial transition period. Forty-five years of use has imbedded PCB's in U.S. industrial practice and it will be costly to replace these unique materials. Alternative dielectric fluids are on the way, but will not be suitable as replacements in existing transformers.

C-2. Remaining uncertainties

The decision by Japan apparently to move further to eliminate these compounds is a real challenge in the public mind to the regulatory policy of the United States. If that Nation can do without PCB's, why cannot we?

Is a total ban worldwide necessary in order to reduce and eliminate contamination to the North American environment? An inventory of PCB's in existence is required and this must be international. We need to know more about the changes in PCB's in use, i.e., dibenzofuran formation. How nondispersive are "closed" electric systems?

We need to reconcile the differences in human food tolerance limits as established by the United States (5 ppm) and Canada (2 ppm) and in alternative proposals from various protagonists on all sides of that question.

Cost/benefit analysis will not be very helpful in the PCB contamination problem because of the great number of subjective judgments and different value systems involved in arriving at a balance. In any event, the marginal costs and the marginal benefits are what must be determined.

We need to know how to remove PCB's from wastewater, i.e., what would be best practicable technology economically achievable in order to get to an effluent standard, and whether that should be 5 ppb, or zero, or what level. We need to know whether 1 ppt is a reasonable ambient water standard in view of the large biomagnification factor.

C-3. Prudent actions

EPA has a responsibility to determine priorities and it needs to review the PCB issue in terms of the total set of pollution problems and the agency's resources. EPA should proceed with effluent standard possibilities in Section 307 of the Water Quality Act (PL 92-500), although such action may end up in court tests. The Endangered Species Act may be a means of cooperative action by other Federal agencies.

Voluntary housekeeping by industry can be increased to a substantial extent, but this requires special attention to the weakest firms, always a problem with voluntary cooperation.

Payment of compensation to fishermen may require an act of Congress similar to the experience with heptachlor in milk some years ago.

It is my personal opinion that the inadvertent and unavoidable addition of PCB materials to foods could be covered by the Delaney Clause if they are found to be carcinogenic in appropriate animal tests.

This conference has addressed a real problem and was not just a demonstration of the prowess of analytical chemistry. However, as with any environmental management issue, the PCB problem must be put in perspective.

PCB's will be in the environment for a long time. Fisheries resources in some of the Great Lakes and rivers have been lost. We can protect against any imminent hazard to human health. Ecosystems are resilient but local damage has occurred. Further leaks from the industrial system must be prevented. Internalizing costs will lead the market to produce replacements for PCB's. Human health is most important but environmental quality is a reflection of the ecosystem that we all share. In this case there may be a coincidence, in that if we protect human health adequately, we will have protected the environment.

All elements of society are in this together and recriminations are not very helpful. Scientists and engineers can generate and deliver information for rational enforcement decisions, searching for equity as the inevitable tradeoffs are made. Verified technical facts and their implications must be communicated efficiently to the public through education and information programs. Most importantly, we must use our ingenuity in solving little parts of the problem when they are all that can be solved as well as in working on an ultimate solution for the problem as a whole.